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THE VITAL CAPACITY OF THE LUNGS OF CHILDREN IN HEALTH AND DISEASE*

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As a result of recent investigations, the clinical value of the determination of vital capacity in diseases of the heart and lungs has attracted attention, especially since this measurement is often found to be greatly reduced by various pathologic conditions of these organs. It has long been recognized, however, that in order for the determination of the vital capacity to be of any great practical value, it is necessary, first, to determine the normal relationship existing between it and other body measurements. Investigators, therefore, have attempted to establish normal standards for vital capacity based on age, height, body weight, stem length, trunk volume and surface area, but in most instances have neglected to determine the extent of normal variability. Since for any body measurement the lung capacity is known to show a certain amount of variability, it is clear that in order to detect abnormal fluctuations, the extent of normal variability must be determined first. Therefore, in connection with the study of vital capacity for children, an attempt is made to estimate the extent of normal variability during childhood. I am indebted to Drs. R. E. Scammon and Wm. Hart for valuable advice concerning the statistical treatment of the data presented in this paper.

1. *Material and Methods.*—Observations are reported for 2,509 normal and healthy children, 1,289 boys and 1,220 girls, ranging in age between 4 and 19 years inclusive. The data (sexes separated) are averaged according to age in Tables 1 and 2, according to standing height in Tables 3, 4, 5 and 6, according to sitting height in Tables 7, 8, 9 and 10, and finally according to body weight in Tables 11 and 12. In addition, observations are recorded for fourteen boys and twenty-six girls (Table 13) afflicted with mitral insufficiency of varying degrees of severity; for seventeen boys and fourteen girls (Table 14) suffering from acute bronchitis; for four boys and five girls (Table 15) with pneumonia; for thirteen boys and four girls (Table 16) subject to asthma; and lastly for seventy-nine boys and seventy-one girls (Tables

* Received for Publication, May 20, 1922.

17 and 18) having tuberculosis. The total of all the observations for vital capacity, including both the normal and also the pathologic cases, amounts to 1,420 with the boys and 1,342 with the girls, a grand total of 2,762 cases.

The vital capacity was measured by means of an accurately calibrated wet spirometer, which offered very little resistance to expiration. The children were carefully instructed to take the deepest possible inspiration, and to empty the lungs completely into the spirometer. As a rule, small groups were taken at a time, so each subject understood from observation how to cooperate to the best advantage. Every child was permitted several trials, and the maximum repeatedly expired was recorded as the vital capacity. The children were urged to do their best, and usually the spirit of competition was sufficiently keen to make one reasonably certain that the records obtained represent the best effort they were capable of exerting. In instances where the cooperation of the child was unsatisfactory, the data were discarded. Furthermore, children having an unusually low vital capacity were included in the averages only in case they gave no history of chronic heart or lung disease. So far as possible all children suffering from acute respiratory infections were excluded from the averages. The vital capacities recorded are not corrected for temperature, aqueous vapor tension, or for variations in barometric pressure.

The children were weighed wearing their ordinary indoor winter clothing. In order to obtain the net weight, the weight of the clothing (computed as recommended by Bardeen[1]) is deducted from the observed gross weight. The standing height (in stocking feet), and also the sitting height (measured with the children seated against a perpendicular scale) was recorded for each child in the majority of instances.

In the analysis of the data for the normal children, as averaged according to age, standing height, and stem length, in each case statistical methods are employed to calculate the degree each average might be expected to fluctuate were observations obtainable upon an infinitely large number of comparable series of cases, and also to determine the extent of absolute and relative variability of vital capacity, by the estimating of the probable error, the standard deviation, and by computing the coefficients of variation, respectively.

The possibility of deriving the approximate normal vital capacity by the estimating of the probable error, the standard deviation, and for any known age, standing height, sitting height, or weight, by means of empirical formula is discussed later on.

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1. Bardeen, C. R.: Weight for Height charts for school boys and girls, Bull. Univ. Wis. Beyer: Growth of U. S. Naval Cadets, Proc. U. S. Naval Inst. **21,** 1895.

2. *Vital Capacity and Age.*—The averages for the vital capacity of the lungs at different ages, as estimated from the nearest birth day, are recorded in Tables 1 and 2. Since there is a sexual difference in vital capacity at corresponding ages, the data for the boys and girls are averaged separately in the tables. In most instances the number of subjects in each group is sufficiently large to render the means fairly significant, especially for the boys from 6 to 16 years, inclusive, and for the girls from 6 to 15 years. For the other groups the averages recorded are probably of less significance, due to the fact that they include relatively fewer observations.

Between 6 and 16 years of age, inclusive, the average vital capacity of the lungs with the boys (Table 1) shows an increase from 1,154 to 3,425 c.c., the absolute gain during the ten years amounting to 2,271 c.c. When expressed in terms of relative increase, this gain amounts to about 197 per cent. Consequently, at 16 years of age the average vital capacity for the boys is practically three times as great as the initial value at 6 years.

With the girls between 6 and 16 years of age, inclusive (Table 2), the average vital capacity increases from 1,085 to 2,778 c.c. The absolute gain in this case amounts to 1,693 c.c., or approximately 578 c.c. less than the increase registered by the boys during a similar period. The relative increase for the ten years amounts to approximately 156 per cent. with the girls, as compared with 197 per cent. gain for the boys. Consequently, at 16 years of age the vital capacity with the girls is only two and one-half times as great as the initial 6 year average.

As is true of physical measurements in general, the vital capacity likewise fluctuates within rather wide limits. Thus, at practically each age the maximum vital capacity recorded (Tables 1 and 2) is two or more times as large as the minimum observed. On inspecting the individual data, however, it is found that in many cases these extreme variations in lung capacity at each age, paralleled to some extent the individual's variation in size from the average of his respective groups. Thus, the unusually low vital capacities recorded, were obtained, as a rule, from children considerably below the average size for their age, whereas the maximum readings were usually for children considerably larger than their group average. The absolute extent of the variation becomes larger as the vital capacity increases, and also is usually greater for the boys than for the girls of corresponding age. Although absolute variability evidently increases with age, and differs for each sex, it does not necessarily follow that relative variability obeys the same rule. In order to determine the degree of relative variability in vital capacity at different ages, and for each sex, it is necessary to resort to methods of statistical analysis of the data. This is attempted in a succeeding paragraph.

TABLE 1.—Normal Boys. Age, Number of Cases, Average Standing Height, Average and Range of Vital Capacity, Mean Vital Capacity Determined by Graphic Interpolation, Absolute and Percentage Annual Increase in Vital Capacity, Formula for Computing Vital Capacity from Age with Value Obtained by Use of Formula, Probable Error, Standard Deviation, and Coefficient of Variation

Age in Years	No. of Cases	Standing Height, Cm.	Average Vital Capacity, C.c.	Minimum Vital Capacity, C.c.	Maximum Vital Capacity, C.c.	Mean V. C. Determined by Graphic Interpolation, C.c.	Absolute Annual Increase in V. C., C.c.	Percentage Yearly Increase in Vital Capacity	V. C. as Computed by Formula $y=(ax)\pm b$ C.c.	Empirical Formula and Values for Constants Used for Each Age Group $y=(ax)\pm b$	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
4	6	103.4	792	500	900	792	...	....	791	$y=$ V. C.			
5	20	106.8	927	600	1,150	958	166	21.0	969	$a=$ Age in years			
6	62	112.2	1,154	800	1,600	1,124	166	17.3	1,146	$x=177.5$	15.6	182	15.8
7	112	116.9	1,290	900	2,200	1,304	180	16.0	1,323	$b=+81$	12.3	194	15.0
8	98	121.8	1,468	1,050	2,100	1,491	187	14.3	1,501		15.0	220	15.0
9	110	129.9	1,715	1,200	2,300	1,679	188	2.6	1,679		15.8	246	14.3
10	87	133.4	1,872	1,400	2,650	1,859	180	10.7	1,856	$y=$ V. C.	18.9	262	14.0
11	113	137.8	1,991	1,300	2,800	2,015	156	8.2	2,034	$a=$ Age in years	17.1	270	13.6
12	114	142.4	2,182	1,300	3,300	2,210	195	9.0	2,212	$x=178.0$ $b=+76$	21.5	340	15.6
13	132	148.7	2,458	1,700	4,000	2,451	241	10.9	2,450	$y=$ V. C.	25.2	430	17.5
14	177	154.8	2,712	1,400	4,300	2,772	321	13.1	2,760	$a=$ Age in years	24.5	484	17.8
15	155	159.9	3,145	1,850	4,400	3,094	322	11.6	3,070	$x=310.0$	29.8	551	17.5
16	67	167.2	3,425	2,100	4,300	3,417	323	10.8	3,380	$b=-1,580$	47.2	573	16.7
17	23	171.4	3,776	2,400	4,500	3,708	289	8.5	3,690				
18	9	172.0	3,922	2,750	4,400	3,999	281	7.6	4,000				
19	4	168.2	4,300	4,100	4,500	4,300	301	7.5	4,310				

TABLE 2.—Normal Girls. Age, Number of Cases, Average Standing Height, Average and Range of Vital Capacity, Mean Vital Capacity Determined by Graphic Interpolation, Absolute and Percentage Annual Increase in Vital Capacity, Formula for Computing Vital Capacity from Age with Values Obtained by Use of Formula, Probable Error, Standard Deviation, and Coefficient of Variation

Age in Years	No. of Cases	Standing Height. Cm.	Average Vital Capacity, C.c.	Minimum Vital Capacity, C.c.	Maximum Vital Capacity, C.c.	Mean V. C. Determined by Graphic Interpolation, C.c.	Absolute Annual Increase in V. C., C.c.	Percentage Yearly Increase in Vital Capacity	V. C. as Computed by Formula $y=(ax)\pm b$ C.c.	Empirical Formula and Values for Constants Used for Each Age Group $y=(ax)\pm b$	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
4	9	95.4	664	350	850	644	...	....	644	y = V. C. in Cm.			
5	26	106.4	888	600	1,200	852	208	32.3	819	a = Age in years			
6	62	111.5	1,085	700	1,600	1,045	193	22.7	995	x = 175.3	14.0	163	15.0
7	81	114.4	1,228	900	1,800	1,215	170	16.3	1,170	b = —57	13.6	181	14.7
8	76	121.0	1,401	800	1,950	1,373	158	13.0	1,345		15.4	199	13.5
9	73	127.0	1,513	1,000	2,250	1,524	151	11.0	1,521		18.1	229	15.1
10	117	132.1	1,672	900	2,400	1,685	161	10.6	1,696		17.0	273	16.3
11	119	135.9	1,799	1,250	2,550	1,871	186	11.0	1,871		14.9	241	13.4
12	135	144.0	2,053	1,400	2,900	2,085	214	11.4	2,085	y = V. C. in Cm.	19.9	343	16.7
13	162	151.4	2,349	1,550	3,600	2,309	224	10.7	2,268	a = Age in years	21.7	409	17.5
14	192	156.6	2,607	1,900	3,800	2,511	202	8.7	2,451	x = 183.0	17.5	361	13.9
15	181	157.8	2,702	1,900	3,700	2,676	165	6.6	2,634	b = —111	24.3	413	15.3
16	29	160.1	2,778	2,050	3,500	2,817	141	5.3	2,817				
17	7	162.6	2,943	2,250	3,400	2,955	138			y = V. C. in Cm.			
18	1	162.0	3,100	.....	.....	3,100	145	4.9	2,958	a = Age in years			
								4.9	3,100	x = 141.2			
										b = +558			

The general trend of the increase in the average vital capacity of the lungs with advancing age is shown graphically in Figure 1. In order to eliminate minor fluctuations the curves have been smoothed by means of graphic interpolation. This produces a curve which probably conforms more exactly with the type of curve that would be obtained from averages based upon an infinitely large number of cases, and probably represents more nearly the true mean vital capacity. The mean vital capacity at each age, as determined by graphic interpolation, is recorded in Tables 1 and 2, and represented in graphic form in Figure 1. The actual averages for the vital capacity are represented in the figure by dots for the boys and by circles for the girls. In general,

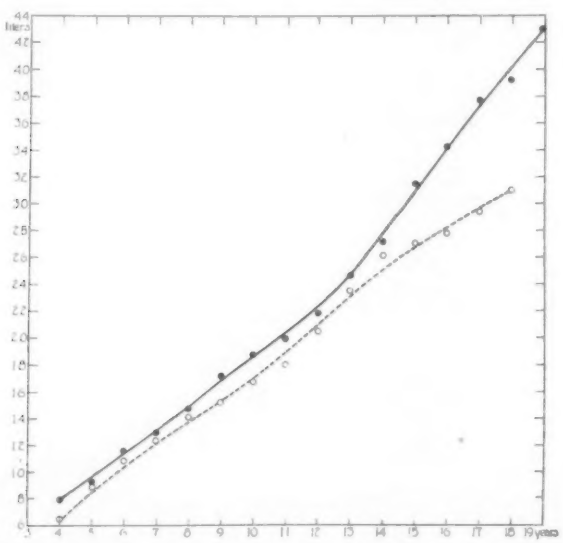

Fig. 1.—Showing smoothed curves representing the average absolute increase in the vital capacity of the lungs as determined by graphic interpolation, for boys between 4 and 19 years of age inclusive, and for girls between 4 and 18 years of age inclusive. The actual averages at each age for the boys are represented by dots and by circles for the girls. Vital capacity in liters is represented on the ordinate, and age in years on the abscissa. The solid line represents the boys; the broken line the girls in this and Figures 2, 3 and 4.

the smoothed curve of vital capacity for each sex tends to assume a concave form corresponding closely with the curve based on the actual averages. From 4 to 11 years, inclusive, the curve for boys is practically a straight line, due to the fair uniformity of the yearly increase in vital capacity. This is followed later by a period of acceleration reaching a maximum at 14 years, which apparently is fairly well maintained, although at a reduced rate, at least until 19. This accounts largely for the general concavity of the curve.

There appear, therefore, to be three main phases in the curve representing the increase in vital capacity for the boys between 4 and 19 years: (1) a period of fairly uniform increase between 4 and 10; (2) a period of acceleration extending from 12 to 14, and (3) a period of uniform but less rapid increase in vital capacity to 19.

The smoothed curve of vital capacity (Fig. 1) for the girls likewise shows several phases. Between 4 and 6 years of age the curve rises rather abruptly. From 7 to 10 the annual increase in vital capacity is fairly uniform but slower than during the earlier period. An acceleration in the rate of increase occurs at 11 and persists until 13. Due, in part, to the failure of this rapid yearly increase in vital capacity to continue during the following five years, the concavity produced by the prepubertal acceleration in the curve of vital capacity as plotted against age is much less marked for the girls than for the boys, and also does not extend over as great a period of years as with the former.

The accelerated increase in vital capacity appearing at 11 and 12 years of age for the girls and boys, respectively, is, no doubt, related to the normal prepubertal acceleration of the growth of the body as a whole.

The difference in vital capacity between girls and boys at corresponding ages, is clearly shown by the curves in Figure 1. Throughout the period studied the boys have the larger lung capacity. At 6 years the difference amounts to approximately 79 c.c., which at 10 years has increased to 174 c.c. Subsequently the girls show a more rapid increase in vital capacity which reduces the difference to 125 c.c. at 12 years of age. From the fourteenth year on, the difference in vital capacity between the two sexes, rapidly increases.

As long ago as 1854 Wintrich [2] noted the existence of a sexual difference in the vital capacity of the lungs, and observed that this difference persists until old age. Since then this observation has been confirmed frequently by practically all investigators who have studied the subject. Among the more recent and extensive observations on children, the data collected by Gilbert,[3] for 1,279 boys and 1,244 girls, and also Smedley's [4] averages for 2,788 boys and 3,471 girls, show the vital capacity at all ages to be larger for the boys. Baldwin [5] apparently

2. Wintrich, M. A.: Einteilung zur Darstellung der Krankheiten der Respirations organe. Virchow, R.: Handbuch der speciellen Pathologie u. Therap. **92,** 1854.

3. Gilbert, J. A.: Researches on the Mental and Physical Development of School Children. Studies from Yale Psychological Laboratory, **2,** 1894. Researches on School Children and College Students. University of Iowa Studies in Psychology. **1,** 1897.

4. Smedley, F. W.: Report of Commissioner of Education **1,** 1902.

5. Baldwin, B. T.: Physical Growth and School Progress, U. S. Bureau of Education Bull. No. 10. The Normal Child. Its Physical Growth and Mental Development, Pop. Sc. Monthly **85,** 1914.

is the only one who has obtained different results. He states that boys have a greater lung capacity on the average than the girls, except between 13 and 14 years of age when just the reverse is true. My own data indicate a larger vital capacity for the boys at all ages, and thus are in agreement with the findings of Gilbert, Smedley and others.

The absolute yearly increase in vital capacity for the boys (Table 1) is fairly uniform until 10 years of age, but at the eleventh year the increase is considerably lower than that registered at any other time. Following this period of retardation, the absolute annual growth in vital capacity becomes accelerated and until 19 years maintains a more rapid rise than occurs at any earlier age.

With the girls the average absolute annual increase in vital capacity (Table 2) also is fairly uniform between 6 and 10 years of age. Starting at 11 years (one year earlier than noted with the boys), the absolute annual increase becomes accelerated, but is maintained only until the fifteenth year; whereas with the boys the vital capacity continues to grow rapidly until 19 years of age. This results on a marked divergence at the period between the curves for vital capacity for the two sexes when plotted against age. The greatest annual gain in vital capacity amounted to 323 c.c. for the boys during the fifteenth year, as compared with a smaller maximum of 224 c.c. for the girls at 13 years of age.

A consideration of the percentage annual increase in vital capacity gives one a conception of the relative rapidity of growth in this respect at different ages. With the boys the rate of relative increase diminishes rapidly from an average annual gain between 4 and 5 years of 21 per cent., to a minimum rate of 8.2 per cent. at 11 years of age. During the following three years, the rate of growth of lung capacity becomes relatively more rapid, rising to 13.1 per cent. at 14 years, but later steadily declines.

With the girls the relative increase in vital capacity decreases from 32.3 to 10.6 per cent. between 5 and 10 years inclusive. During the following two years the rate of annual growth becomes more rapid, amounting to 11.4 per cent. at 12 years as compared with a rise to 13.1 per cent. for the boys at 14. Following this period of acceleration, the rate of increase in lung capacity diminishes rapidly.

In general, then, for each sex the relative rate of annual growth in vital capacity shows a tendency to decrease during early childhood, becomes slightly more rapid about the time of the advent of puberty, and later gradually declines. The period of acceleration in the rate of lung capacity growth begins and terminates at an earlier age with the girls than with the boys.

The data published by Smedley,[4] which are the most extensive series of observations on children at present available in the literature, like-

wise show that the rate of annual increase in vital capacity diminishes during early childhood, rises about the time of puberty and later declines. His averages also indicate that the prepubertal acceleration appears later, and is relatively greater with the boys than with the girls.

If we now return to a consideration of the smoothed curves of vital capacity as plotted against age (Fig. 1) it is found possible to divide the curve for each sex into three phases, so selected that each segment conforms closely to straight lines of different incliniation. Thus for the boys the portions of the curve between 4 and 9 years, between 10 and 12 years, and between 13 and 19 years deviate but slightly from the straight lines connecting these various points. Likewise, with the girls the successive segments from 4 to 11, 12 to 14, and 15 to 18 years of age, inclusive, also conform rather closely to the different straight lines drawn through these points.

The value of any point upon a straight line may be readily determined by means of the empirical formula $y = ax \pm b$, where $y =$ the unknown value, $a =$ a known variable, $x =$ a constant, and $b =$ a second constant. It follows, therefore, that the vital capacity between successive segments of the curve conforming closely to a straight line may be determined in a fairly satisfactory and accurate manner by employing the above formula where $y =$ the vital capacity to c.c., $a =$ the age in years, $x =$ a constant, and $\pm b =$ a second constant. The vital capacity as derived by formula, and also the different values for the constants x and b are given in Tables 1 and 2. On inspecting the data the computed vital capacities are found to agree rather closely with the means as determined by graphic interpolation. With the boys the greatest difference occurs at 16 years where value derived by formula is 37 c.c. too low. For the girls the greatest deviation amounts to 60 c.c. at 14 years. Evidently, therefore, the vital capacity of the lungs for each between certain ages, may be derived with fair accuracy by means of a simple straight line formula.

The necessity of establishing some relationship between the vital capacity of the lungs and other body measurements in order to be of any practical clinical value has long been recognized. In 1846 Hutchinson,[6] by inventing the spirometer, was the first to obtain extensive and accurate records of vital capacity. From this scientific pioneer study Hutchinson concluded that a definite relationship exists to standing height, the vital capacity increasing in a simple arithmetical progression with increase in height. For each additional inch with individuals between 5 and 6 feet tall he states that 8 additional cubic inches of air at 60 F. may be given out by a forced expiration. Hutchinson also

6. Hutchinson, J.: On the Capacity of the Lungs and on the Respiratory Functions, Med. chir. Tr. **29,** 1846, Lancet **1**· 1846.

recognized that weight, age and sex might modify averages to some extent, but believed, however, that no definite relationship existed between vital capacity and either the body weight, the sitting height, or the chest circumference.

Fabius [7] pointed out the occurrence of marked individual variation from averages based on height. He asserted that the vital capacity divided by the product of of the stem length (measured from the external accipital protuberance to the tip of the coccyx) and the chest circumference produced a constant which varied only a slight extent with different individuals. The cause of this variability in the constant Fabius attributed to differences in age and chest expansion. Fabius emphasized, therefore, that the length, circumference and mobility of the chest should be measured in determining whether or not an individual's vital capacity is normal.

Wintrich [8] investigated the vital capacity of the lungs of 3,500 healthy persons including 500 women. He noted that during childhood age influenced the vital capacity markedly, and also that a sexual difference persisted into old age. Wintrich was interested mainly in the relationship between vital capacity and height.

Schneevoogt [8] concluded that standards of vital capacity in relation to age and height are of the most practical value, and claimed that deviations of 500 c.c. from such standards could not be assumed to be necessarily abnormal.

Arnold [9] found extremes of fluctuations to be greater when comparisons were made on a basis of stem length, rather than on standing height. In discussing the smaller vital capacity of the lungs of women as compared with the men relative to height and chest circumference, Arnold points out that the chest expansion is less for the females than for the males.

From a study of the vital capacity of the lungs of 515 students, Kottelman [10] observed that the lung capacity increases with age faster than the growth in body length occurs. MacDonald [11] points out that the data presented by Kottelman also show that weight increases with age more rapidly than increase in lung capacity.

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7. Fabius, H.: De spirometro ejusque usu observationibus cum alioum, tum propriis illustrato, Diss. Amsterdam, Ztschr. rationell. Med. **4,** 1853.

8. Schneevoogt, G. E. V.: Ztschr. rationelle med. **9,** 1854, (cited by Lungsgaard and Van Slyke).

9. Arnold, F.: Ueber die Athmungsgrösse des Menschen, Heidelberg, 1855.

10. Kottelman, L.: Die Körper verhält nisse der gelehrten Schüler des Johannaens in Hamburg, Ztschr. d. Königlch Preussischen statistischen Bureaus, Berlin, 1879.

11. Mac Donald, A.: Experimental Study of Children, U. S. Bureau of Education Report of Commisioner **1,** 1897.

The existence of a relationship between vital capacity and weight is disputed by Hall.[12] This observer arrived at the unique conclusion that the vital capacity varies directly with the strength of the individual.

Bohr [13] objected to the use of standing height for estimating the normal vital capacity on the grounds that the individual variability is so great as to render this method inapplicable. He maintained that the total and residual air as well as the vital capacity should be determined, and placed greater emphasis upon the percentage relationship between these various subdivisions of the total lung volume, than on their absolute value. Hasselbach,[13a] Rubow,[14] Siebeck,[15] Bie and Maar [16] and Plesch [17] also investigated the relationship between the total lung volume, the middle and vital capacities and the residual air in normal and pathologic conditions, and likewise were interested more in the relative than in the absolute value of the different volumes.

Peabody and Wentworth,[18] studying the vital capacities for a group of 140 healthy adults (ninety-six males and forty-four females), divided the subjects into three groups according to their height and considered the average vital capacity for each group as a normal standard for corresponding height. Of the ninety-six men, 84 per cent. registered vital capacities falling within 10 per cent. of their respective normal. These same authors, in a study of a small group of cases, have also found a rather close relationship to the body surface area.

Recently Lundsgaard and Van Slyke [19] have claimed that the vital capacity of the lungs is closely related to the calculated chest volume.

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12. Hall, W. S.: Changes in the Proportions of the Human Body During the Period of Growth, J. Authropol. Inst. of Great Britain and Ireland **25,** 1896.

13. Bohr, C.: Die funktionellen Aenderungen in der Mittellage und Vital Kapazität der Lungen, Deutsch. Arch. f. klin. Med. **88,** 1906.

13*a*. Hasselbach, K. A.: Ueber die Einwinkung der Temperatur auf die vitale Mittelage der Lungen. Ueber die Total Kapazität der Lungen, Deutsch. Arch. f. klin. med. **93,** 1908.

14. Rubow, V.: Untersuchungen über die Atmung bei Herzkrankheiten; ein Beitrag zum Studium der Pathologie des kleinen Kreuslaufes, Deutsch. Arch. f. Klin. Med. **92,** 1908.

15. Siebeck, R.: Ueber die Beinflussung der Atemmechanik durch krankhafte Zustande des Respirations und Kreislaufapparates, Deutsch. Arch. f. klin. Med. **100,** 1910.

16. Bie, W., and Marr, W.: Dem Fall von angeborenem Herzfehler mit Cyanose und Polyglobulie, Deutsch. Arch. f. klin. Med. **99,** 1910.

17. Plesch, J.: Die pathologische Physiologie des Lungen volumens und seine Beziehung zum Kreislauf, Ztschr. f. Exper. Path. u. Therap. **13:** 1913.

18. Peabody, F. W., and Wentworth, J. A.: Clinical Studies on Respiration. IV. The Vital Capacity of the Lungs and Its Relation to Dyspnea, Arch. Int. Med. **20:**468 (Sept.) 1917. Peabody, F. W.: Cardiac Dyspnea, Am. J. M. Sc. **155:**100, 1918.

19. Lundsgaard, C., and Van Slyke, D. D.: Studies of Lung Volume 1. Relation Between Thorax Size and Lung Volume in Normal Adults, J. Exper. M. **27:**651, 1918.

The chest they considered as a geometrical figure and the product of three dimensions was considered to represent a volume proportional to the chest volume. The height of the chest was taken as the length of the sternum from the superior border to a point just below the articulation with the xiphoid cartilage. The depth was taken as the horizontal distance from the middle of the sternum at the level of the insertion of the third costal cartilage, to the spinal column; and the breadth as the distance across the sixth rib in the mid axillary line.

The normal vital capacity in man and its relation to the size of the body has also been the subject of a paper published recently by Dreyer.[20] Based largely on the data of Hutchinson[6] and Schuster,[21] together with some observations of his own, Dreyer has claimed that the vital capacity is a function of the weight. This, he says, can be expressed by the formula $\frac{W^n}{v.c.} = K$, where W is the net weight in grams, v.c. the vital capacity in cubic centimeters, the power n is 0.72, and K is a constant. He further states that since it is already established that $\frac{W^n}{S} = K_2$ where W = net weight, S = body surface and n is 0.72, it follows that the vital capacity is a simple function of the body surface.

The relation between the vital capacity and stem length Dreyer finds to be correctly expressed by the formula $\frac{SH^2}{v.c.} = K_3$ where SH = the stem length in centimeters, v.c. = vital capacity in cubic centimeters, the power n is approximately 2, and K_3 is a constant. Between the vital capacity and the chest circumference the existing relation can be expressed by the formula $\frac{Ch^n}{v.c.} = K_4$, where Ch = circumference of chest in cubic centimeters, v.c. = the vital capacity in centimeters, the power n is approximately 2, and K_4 is a constant.

Also $\frac{SH \times Ch}{v.c.} = K_5$ where SH stem length in centimeters Ch = chest circumference in centimeters v.c. = vital capacity in cubic centimeters, and K_5 is a constant. Dreyer has pointed out, therefore, that the vital capacity is definitely related to the 0.72 power of the weight, directly to the body surface area, to the square of the stem length, to the square of the chest circumference, and to the trunk volume (estimated as the product of the chest circumference and stem length), and states that the above is also the order of the importance of these relations.

In discussing the variability of vital capacity in normal persons, Dreyer shows that for the 16 individuals which he studied the mean deviation, as estimated by the method of least squares, of the values

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20. Dreyer, G.: Investigations on the Normal Vital Capacity in Man and its Relation to the Size of the Body, Lancet **2**:227, 1919. Dreyer, G., and Burrell, L. S. T.: The Vital Capacity Constants Applied to the Study of Pulmonary Tuberculosis, Lancet **1**:1212, 1920.

21. Schuster, E.: First results from the Oxford Authropometric Laboratory, Biometrika **8**, 1911.

for K computed from the formula based on the two thirds and the 0.72 power of the body weight, in the two cases amounted to 3.56 and 2.64 per cent., respectively, or distinctly lower when the best power for the weight (0.72) is used. This, Dreyer says, indicates that if a person is found to have a vital capacity 6 per cent. smaller or larger than that calculated from his body surface, he probably has a normal vital capacity, and if the vital capacity is 10 per cent. above or below the normal it is almost certain to be abnormal. On using the data of Fitzgerald, Purefoy and Haldane,[22] however, a mean deviation of 15.8 per cent. is obtained, or five times as great as in Dreyer's series. Dreyer attributes this great difference in variability to the fact that the observations of Fitzgerald, Purefoy and Haldane were carried out on a more highly mixed material. He also calls attention to the differences in vital capacity exhibited by the different classes studied by Hutchinson, which apparently have nothing to do with fundamental bodily deficiencies but are simply a result of conditions depending upon occupation and mode of life.

The existence of normal individual difference of vital capacity has constituted a real difficulty in its practical clinical application, but aside from this study of Dreyer's, apparently no one has attempted to estimate its extent by statistical methods. It is absolutely essential to determine the magnitude of the deviation from the average met with in normal and healthy individuals, before it is possible to decide whether the vital capacity for any given person should be considered normal or abnormal. Knowing the extent of normal variability of vital capacity also enables one to more fully appreciate both the limitations and the possibilities of its applicability in clinical work. In this study an attempt is made to determine the extent of variability of vital capacity during childhood.

Before entering into a consideration of the normal variability, a consideration of the reliability of the averages obtained for the vital capacity seems appropriate. The measure of the trustworthiness of an average derived from a limited number of observations is furnished by the so-called probable error of random sampling. To be more explicit, the probable error of random sampling is a measure of the extent that the average values would be expected to vary, were the observations made at random upon an infinitely large number of comparable series of cases, and is so chosen, that half of these values would lie inside, and half outside the limits of the probable error. Therefore in an infinitely large series roughly 50 per cent. of the averages will fall within the limit of the range of the probable error about the average based upon a limited number of cases. The probable error assumes

22. Fitzgerald, Purefoy, M., and Haldane, J. S.: The Normal Alveolar Carbonic Acid Pressure in Man, J. Physiol. **32**, 1905.

the data to be technically correct, and has nothing to do with inaccuracies in measurement. The probable error of the average or mean vital capacity at each age has been calculated by means of the formula $0.6745 \times \frac{S}{\sqrt{n}}$ where S = standard deviation, and n = number of observations, and the results obtained may be found in Tables 1 and 2. The derivation and significance of the standard deviation is discussed at a later time.

During childhood it appears that in general, for the various groups, the calculated probable error of random sampling for each sex (Tables 1 and 2) constantly increases with advancing age. For the boys the value 15.6 c.c. at 6 years has increased to 29.8 c.c. at 15 years of age. With the girls during this same period the probable error increases from 14 at 6 to 24.3 at 15 years. In applying this information we can conclude for the boys at 15 years of age, that were the vital capacity taken for a large series of comparable groups of individuals the chances are that half these series would show averages falling between 3,145 c.c. ± 29.8 c.c., while the remaining 50 per cent. lie either above or below this limit. In other words, for boys at this age, 50 per cent. of all group averages can be expected to vary not more than 29.8 c.c. above or 29.8 c.c. below that obtained. Derivations markedly beyond these limits probably indicate the existence of biological differences between the groups concerned.

The most commonly used measure of the dispersal of the individual measurements from the average of the sum of all the observations is the standard deviation. The standard deviation is obtained first by squaring all the deviations from the average, summing these squares, and then dividing this sum by the number of deviants, which gives the mean-square deviation. The square root of this mean-square deviation is designated as the standard deviation. The practical value of the standard deviation lies in the fact that it indicates the degree of the absolute variability, within the limits of which the probability is that the measurement obtained is more apt to be normal, and commonly the greater the deviation beyond the limits established by the standard deviation the greater the probability is that the measurement is abnormal. It is also particularly useful in the determination of measures of interrelationship.

The standard deviation from the average vital capacity of the lungs at each age has been calculated for the boys between 6 to 16 years, inclusive, and from 6 to 15 years for the girls (Tables 1 and 2). The number of subjects in each group during this period was considered sufficiently large to justify the procedure. Between 6 and 16 years of age for both sexes the absolute value of the standard deviation increases roughly three fold. With the boys it increases from about 182 c.c. at

6 years to 573 c.c. at 16 years. If we attempt to apply this information in considering whether or not any single observation falls within normal limits, then at 16 years of age the vital capacity in individual cases might vary 573 c.c. either above or below the average value of 3,425 c.c. at this age, and could not be considered as definitely abnormal. In clinical work, however, those subjects approaching the lower limits of normal variability would, no doubt, cause the greater concern.

With the girls the standard deviation (Table 2) increases from 163 c.c. at 6 years to 413 c.c. at 15 years. At corresponding ages the absolute value of the standard deviations are usually somewhat smaller for the girls than for the boys.

In order to make comparison between the relative degree of variability of the vital capacity at different ages, it is necessary to reduce the various standard deviations to a comparable basis. This is readily accomplished by computing the percentage that the standard deviation at each age forms of the average vital capacity for that period. This relative variability constant is known as the coefficient of variation, and is in reality the per cent. that the standard deviation forms of the respective mean or average.

For the boys the coefficient of variation (Table 1) between 6 and 11 years of age apparently decreases from 15.8 to 13.6. Thus at 6 years the normal variability of vital capacity expected is approximately 15.8 per cent. above or below the average for that age, which at 11 years has decreased to 13.6 per cent. During the following years the vital capacity becomes considerably more variable, the coefficient of variation at 14 years rising to 17.8, with apparently a tendency to decrease during the succeeding two years.

For the girls the coefficient of variation (Table 2) decreases from 15 at 6 years to 13.5 at 8 years. Subsequently it rises, reaching a maximum of 17.5 at 13 years, one year earlier than the maximum coefficient of variation recorded for the boys. Later the variability apparently decreases. At 11 years of age an unusually low coefficient of 13.4 was obtained from the data, which possibly may be accidental in character. On consideration of the extent of variability in vital capacity it appears that for each sex variability decreases during early childhood, increases about the period of puberty, and later declines. In the main essentials the general trend of the variability in vital capacity is quite similar to that noted in respect to variability in weight and height during growth, for similarly the variability of these two physical measurements decreases at first, rises to attain a maximum about the time of puberty, and then diminishes again.

Although the standard deviation and the coefficient of variation gives a rough measure of the degree of absolute variability, a further conception of variability may be gained through the determination of the

probable error of the data. Provided the curve of distribution of the individual observations conforms to the ideal mathematical type of symmetrical distribution curve, the statistical constant known as the probable error of the data establishes limits within which approximately 50 per cent. of all observations taken at random will be expected to fall. Of the remaining 50 per cent. of observations, 25 per cent. will lie above and 25 per cent. below the limits established. The probable error of the data may be computed by means of the formula $0.6745 \times SD$, where SD is the standard deviation. In general, if a large number of observations is collected, one finds that the majority occur near the average value for the measurement taken, and the greater the deviation from the average the smaller the groups become. The frequency or distribution curve plotted for extensive series of observations often is found to conform very closely to the ideal symmetrical mathematical type of distribution or probability curve. Under such circumstances the determination of the probable error of the data of this series of observations is of especial significance as explained above. With respect to the observations on the vital capacity of the lungs the value and significance of the distribution curves based upon this data is greatly limited due to the small number of observations available. The data at present are insufficient to establish definitely the character of their distribution curve. At some later time it is hoped to be able to accumulate enough data to definitely establish an approximate limiting distribution curve for this measurement. To date, however, the curves obtained for the vital capacity in general appear to conform fairly well with the ideal symmetrical mathematical distribution curve. For the present, therefore, I feel it safe to assume that approximately 50 per cent. of cases taken at random will have vital capacities falling within the limits established by the probable error of the standard deviation for the group concerned. Twenty-five per cent. will lie above and 25 per cent. below these limits. If expressed in terms of per cent., the children constituting the last group, in general, have vital capacities 10 per cent. or more below the expected normal. By recording any individual vital capacity, one thus may easily classify the patient as to the group to which the child belongs. Subjects falling into the group representing the poorer one fourth of the child population are children who probably deserve special study to eliminate pulmonary or cardiac disease as a factor causing their low standard of physical fitness. Experience has shown, however, that an apparently normal vital capacity does not exclude the necessity of thorough examination when the patient is suspected of having tuberculosis, for occasionally the vital capacity may be normal as compared with averages, even in the presence of serious disease of the lungs. Should the accumulation of more extensive observation on the vital capacity of the lungs reveal the fact

that the frequency curve derived from such data does not conform to the ideal symmetrical mathematical probability curve, but shows a certain definite skewness the above statements necessarily must be modified.

3. *Vital Capacity and Standing Height.*—In Tables 3 and 4 the data have been averaged in groups (sexes separated), each group including subjects differing 5 cm. or less in standing height, age and other physical measurement being disregarded. With a growth from an average of 108 cm. to 168 cm. in height, the vital capacity for the boys (Table 4) has increased from 1,074 c.c. to 3,625 c.c., an increase of 2,551 c.c. Thus, with an increase of 60 cm. in standing height representing approximately 54.5 per cent. growth of this measurement, the vital capacity has increased more than 237 per cent. On the whole, then, growth of vital capacity at this period with the boys, occurs about four times as rapidly as the growth in height.

For the girls, accompanying a growth in height from 108 cm. to 168 cm. the vital capacity (Table 4) increases from 1,022 c.c. to 3,017 c.c. This increase of 1,995 c.c. represents a percentage increase of about 195 per cent., as compared with 237 per cent. with the boys for a corresponding growth in height. Nevertheless, also with the girls the lung capacity increases more rapidly than increase in height occurs.

The increase in lung capacity for each sex as plotted against standing height is represented graphically by means of smoothed curves (as determined by graphic interpolation) in Figure 2. The curves for each sex when determined by this method tend to pursue a sinuous course, being somewhat concave in form with the boys up until the standing height reaches approximately 175 cm., and with the girls until they become about 165 cm. tall. Beyond these points the direction of each curve changes tending to straighten out to some extent. The greater vital capacity for the boys as compared with the girls of corresponding height is clearly shown. The extent of the difference between the boys and girls in general appears to increase constantly as they grow taller. If we analyze the curves more completely we find that with the boys the increase in vital capacity plotted against height, is fairly uniform until the individuals are about 148 cm. tall. With growth from 148 cm. to 173 cm. in height, the increase in vital capacity becomes more rapid, but beyond 173 cm. it diminishes considerably in rate.

For the girls the most rapid increase in vital capacity accompanies the growth in height from 143 to 160 cm.

The absolute increase in vital capacity (Tables 3 and 4) which accompanies each successive growth of 5 cm. in standing height is fairly uniform with the boys until they become approximately 143 cm. tall and with the girls until they reach a standing height of 138 cm. At

TABLE 3.—Normal Boys. Number of Cases, Average Standing Height, Average Vital Capacity, Mean Vital Capacity as Determined by Graphic Interpolation, Absolute and Percentage Increase in Vital Capacity Accompanying Each 5 Cm. Growth in Height, Vital Capacity as Computed from Height by Formula for Each Height Group, Probable Error, Standard Deviation, and Coefficient of Variation

Number of Cases	Standing Height, Cm.	Average Vital Capacity, C.c.	Mean V. C. Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity, C.c.	Percentage Increase in Vital Capacity, C.c.	Vital Capacity as Computed by Formula $y=(ax)\pm b$	Empirical Formula for Each Height Group $y=(ax)\pm b$	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
3	98	837	837	...	....	838	y = V. C.			
6	103	788	912	75	9.0	963	a = Height in Cm.			
40	108	1,074	1,062	150	16.4	1,089	x = 25.1	15.0	140	13.0
83	113	1,190	1,194	132	12.4	1,214	b = —1,622	11.0	149	12.5
84	118	1,332	1,340	146	12.2	1,340		11.1	151	11.8
89	123	1,474	1,500	160	11.9	1,499	y = V. C.	12.6	176	11.9
96	128	1,690	1,674	174	11.6	1,684	a = Height in Cm.	13.6	197	11.7
109	133	1,828	1,853	179	10.7	1,869	x = 37	12.2	188	10.3
118	138	2,050	2,034	181	9.8	2,054	b = —3,052	14.0	227	11.1
123	143	2,230	2,220	186	9.1	2,239		15.5	255	11.4
114	148	2,359	2,423	203	9.1	2,424		15.8	278	11.8
97	153	2,594	2,660	237	9.8	2,659	y = V. C.	18.1	264	10.2
115	158	2,936	2,935	255	9.5	2,948	a = Height in Cm.	22.4	357	12.2
75	163	3,173	3,243	308	10.5	3,237	x = 57.8	29.1	373	11.7
77	168	3,625	3,547	304	9.4	3,526	b = —6,184	31.7	413	11.5
35	173	3,919	3,815	268	7.6	3,815				
16	178	4,094	4,020	205	5.4	4,020	y = V. C.			
3	183	4,167	4,175	155	3.9	4,160	a = Height in Cm.			
1	188	4,300	4,300	125	3.0	4,300	x = 28 b = —964			

TABLE 4.—Normal Girls. Number of Cases, Average Standing Height, Average Vital Capacity, Mean Vital Capacity as Determined by Graphic Interpolation, Absolute and Percentage Increase in Vital Capacity Accompanying Each 5 Cm. Growth in Height, Vital Capacity as Computed from Height by Formula, Formula for Each Height Group, Probable Error, Standard Deviation, and Coefficient of Variation

Number of Cases	Standing Height, Cm.	Average Vital Capacity, C.c.	Mean V. C. Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity, C.c.	Percentage Increase in Vital Capacity with 5 Cm. Growth in Length, C.c.	Vital Capacity as Computed by Formula $y=(ax)\pm b$	Empirical Formula for Each Height Group $y=(ax)\pm b$	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
5	98	755	755	...	....	755	(25.5 × Ht.) — 1,744 = V. C.			
20	103	851	876	121	16.0	882				
37	108	1,022	1,005	129	14.7	1,010				
68	113	1,118	1,136	131	13.0	1,137		11.9	146	13.1
71	118	1,283	1,276	140	12.3	1,276	(29.5 × Ht.) — 2,205 = V. C.	12.2	153	11.9
86	123	1,412	1,414	138	10 8	1,423		10.6	146	10.3
85	128	1,555	1,560	146	10.3	1,571		12.7	173	11.1
93	133	1,698	1,707	147	9.4	1,718		12.9	185	10.9
104	138	1,870	1,865	158	9.3	1,866		13.4	208	10.9
109	143	2,005	2,036	171	9.2	2,036	(39.7 × Ht.) — 3,641 = V. C.	14.8	229	11.4
98	148	2,211	2,230	194	9.5	2,234		17.3	254	11.5
164	153	2,449	2,439	209	9.4	2,432		16.4	312	12.7
141	158	2,661	2,646	207	8.5	2,631		18.6	328	12.3
96	163	2,855	2,830	184	7.0	2,830		22.8	332	11.6
35	168	3,017	2,978	148	5.2	2,978	(24.3 × Ht.) — 1,104 = C. C.			
3	173	3,100	3,100	122	4.1	3,100				

these points the absolute increase rises rapidly reaching a maximum of 308 c.c. for the boys when 163 cm. tall, as compared with 209 c.c. for the girls at a standing height of 153 cm. Subsequently the absolute increase for each sex steadily declines. Evidently, therefore, with each sex the increase in vital capacity for each 5 cm. growth in height is not uniform throughout childhood. Hutchinson's conclusion, based on data collected largely from adults, that vital capacity increases in a simple arithmetical progression with increasing height certainly does not apply to the period of childhood. Dreyer has demonstrated recently from Hutchinson's data that this contention was erroneous, and has shown that even for adults the vital capacity is not a simple function of the body height, since it increases in an irregular manner and not at a fixed rate with each unit increase in height.

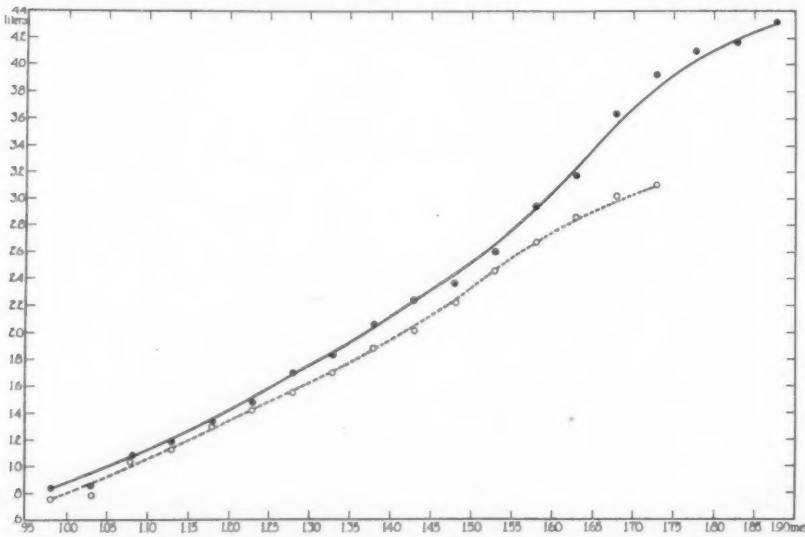

Fig. 2.—Showing smoothed curves representing the average absolute increase in the vital capacity of the lungs as determined by graphic interpolation, for boys between 0.98 and 1.88 meters tall inclusive, and for girls between 0.98 and 1.73 meters tall inclusive. The actual observed averages at each standing height are represented by dots for boys, and by circles for the girls. Vital capacity in liters is represented on the ordinate, and standing height in meters on the abscissa.

The relative increase in vital capacity accompanying each 5 cm. growth in standing height (Tables 3 and 4) apparently diminishes throughout childhood. There is a slight indication of an increase in percentage growth in vital capacity for the boys between 153 and 168 cm. standing height, and for the girls from 148 to 153 cm. tall. The slight tendency to acceleration in rate after an earlier period of decline is much less striking in the case of the averages based on standing height, as compared with that based on age.

As in the case of the curves for vital capacity as plotted against age, likewise those plotted against standing height may be divided into several phases so selected that each phase conforms very closely to a straight line. The empirical formula $y = (ax) \pm b$ by which each of these straight lines may be derived, therefore, affords a fairly satisfactory method by which the vital capacity at any height may be computed when $y =$ the vital capacity, $a =$ the standing height in cm., $x =$ a constant, and $\pm b =$ a second constant. The vital capacities, as computed by means of the above formula, are recorded in Tables 3 and 4, and, in general, agree fairly closely with the averages as determined by means of graphic interpolation. The constants used in computing the values for each portion of the two curves may be obtained by referring to Tables 3 and 4.

The probable error of random sampling (Tables 3 and 4) computed for the vital capacities as averaged according to standing height, in general, with each sex tends to become larger as the individuals grow taller and have a greater lung capacity. For all groups, however, the probable error is comparatively small.

The standard deviations about the means based on standing height (Tables 3 and 4) for each sex tend to increase as the groups become taller, and are, as a rule, greater for the boys than for the girls of corresponding height. In general, the absolute values obtained for the standard deviation seem to be somewhat greater when the data are averaged in groups differing one year in age, as compared with the deviation occurring when the subjects are averaged in groups differing 5 cm. in standing height.

On reducing the standard deviation to the coefficient of variation (Tables 3 and 4) one finds the vital capacity from each sex to become relatively less variable with increasing height until they become 133 cm. tall. Subsequently the variability increases 2 per cent. for a short period and later declines. The general trend of relative variability in vital capacity with relation to average based upon either age or height are quite similar, in both instances showing a tendency to decrease at first, followed by a period of increased variability, which later declines again. The actual values for the coefficients of variation (Tables 3 and 4), for the most part, vary between 10.5 and 12.5. It appears, therefore, that individual records of vital capacity fluctuating between 12 per cent. above, or 12 per cent. below the average expected for corresponding height, could not be interpreted as definitely abnormal. Should the record vary more than 12 per cent. below the averages, the individual record immediately falls into the class that are apt to be abnormal, and such persons, no doubt, should be examined with care to determine the cause of their low standard of physicla fitness in this respect.

If the coefficients of variation for the averages based on age are compared with those derived from averages based on height, the values will be found to be considerably higher in the former case. Due to the presence of less variability, it appears, therefore, that averages based on grouping of the subjects differing 5 cm. in standing height, for more satisfactory normal standards, than the averages based on age at yearly intervals.

By distributing and averaging the cases at each age according to height, as accomplished in Tables 5 and 6, one is able to gain some conception of the influence of these two factors on the vital capacity. When arranged in this manner it is plainly evident that at any age the taller subjects tend to have a much larger vital capacity than the shorter ones of the same age. This holds true for the girls as well as for the boys. The difference is quite considerable in many instances. Thus, for example, at 14 years of age the average vital capacity for twenty-five girls approximately 150 cm. tall was 2,292 c.c., whereas for thirty-five girls of the same age but approximately 15 cm. taller, the vital capacity averaged 2,824 c.c. or 532 more than for the shorter group. The condition of other ages is similar. It is evident, therefore, that at any age differences in standing height influence the vital capacity to a considerable degree. If we now consider the influence of age on the vital capacity for individuals of about the same standing height no such striking difference is found. In several instances the average vital capacity is slightly greater for the older than for the younger individuals of corresponding average height, but this is not consistently the case. On the whole, it seems clear that the difference in the vital capacity of the lungs among children of the same age but differing considerably in height, is far more striking than the differences noted among children of the same height but of different age. However, in comparing individual records with normal standards it is probably advisable to take both age and height into consideration.

4. *Vital Capacity and Sitting Height.*—In Tables 7 and 8 the data for each sex have been averaged in groups, each group including subjects differing 2 cm. or less in sitting height.

For the boys the averages (Table 7) indicate that with an increase in stem length from 57 to 87 cm. the vital capacity increases from 1,071 to 3,607 c.c. Hence, with a growth of 30 cm. in stem length, the vital capacity shows an average increase of 2,536 c.c., or nearly 237 per cent.

With the girls (Table 8) for a similar growth in sitting height, the vital capacity increases from an average of 908 c.c. at 57 cm. stem length to 2,967 c.c. at 87 cm. stem length. The percentage increase in this case amounts to 197 per cent., or approximately 40 per cent. less than the increase registered by the boys for a similar growth in

TABLE 5.—Vital Capacity, in Cubic Centimeters, of Normal Boys, for Each 5 Cm. Difference in Standing Height at Each Age, and Average Vital Capacity for Each 5 Cm. of Growth in Height, Combining All Ages

Age in Years	Standing Height																		
	98	103	108	113	118	123	128	133	138	143	148	153	158	163	168	173	178	183	188
5	837	767	978	975	925	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
6	.....	808	1,122	1,150	1,245	1,333	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
7	.....	.....	1,079	1,215	1,308	1,396	1,620	.....	2,200	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
8	.....	.....	.....	1,242	1,355	1.480	1,707	1,650	1,800	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
9	.....	.....	.....	1,400	1,450	1,537	1,672	1,794	1,992	2,075	.....	.....	.....	.....	.....	.....	.....	.....	.....
10	.....	.....	.....	.....	1,525	1,660	1,662	1,872	2,001	2,227	1,900	.....	.....	.....	.....	.....	.....	.....	.....
11	.....	.....	.....	.....	.....	1,463	1,808	1,827	2,065	2,150	2,184	.....	.....	.....	.....	.....	.....	.....	.....
12	.....	.....	.....	.....	.....	1,300	1,575	1.800	2,059	2,151	2,323	2,655	2,975	3,000	.....	.....	.....	.....	.....
13	.....	.....	.....	.....	.....	.....	.....	1,908	2,132	2,244	2,378	2,546	2,768	3,281	3,400	.....	.....	.....	.....
14	.....	.....	.....	.....	.....	.....	.....	.....	1,938	2,298	2,400	2,584	2,906	3,022	3,328	3,538	3,950	.....	.....
15	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,445	2,565	2,727	2,971	3,200	3,673	3,893	3,862	.....	.....
16	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,400	2,400	2,400	3,236	3,205	3,644	3,958	4,180	.....	.....
17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,833	2,850	3,813	3,973	4,262	4,050	4,300
18	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,750	.....	3,925	4,125	4,200	4,400	.....
19	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	4,300	4,300	4,300	.....	.....	.....
Absolute increased vital capacity	837	788	1,074	1,190	1,332	1,474	1,690	1,828	2,050	2,230	2,359	2,594	2,936	3,173	3,625	3,919	4,094	4,167	4,300

TABLE 6.—Vital Capacity, in Cubic Centimeters, of Girls, for Each 5 Cm. Difference in Standing Height at Each Age, and Average Vital Capacity for Each 5 Cm. of Growth in Height, Combining All Ages

Age in Years	Standing Height																	
	88	95	98	103	108	113	118	123	128	133	138	143	148	153	158	163	168	173
4	612	650	692	850	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
5	.....	.....	850	850	914	1,050	1,100	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
6	.....	.....	.....	806	1,034	1,082	1,283	1,317	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
7	.....	.....	.....	950	1,094	1,131	1,239	1,356	1,440	1,800	.....	.....	.....	.....	.....	.....	.....	.....
8	.....	.....	.....	900	1,000	1,231	1.311	1,444	1,455	1,780	.....	.....	.....	.....	.....	.....	.....	.....
9	.....	.....	.....	.....	.....	1,150	1,294	1,430	1.509	1,687	1,820	1,850	.....	.....	.....	.....	.....	.....
10	.....	.....	.....	.....	.....	1,075	1,333	1,423	1,588	1,684	1,821	1,980	2,183	2,150	.....	.....	.....	.....
11	.....	.....	.....	.....	.....	.....	1,325	1.342	1,632	1,703	1,820	1,943	2,143	2,050	.....	.....	.....	.....
12	.....	.....	.....	.....	.....	.....	.....	1,450	1,725	1,700	1,925	1,996	2,110	2,352	2,533	2,950	.....	.....
13	.....	.....	.....	.....	.....	.....	.....	.....	1,650	1,650	1,957	2,020	2,257	2,381	2,515	2,865	3 040	.....
14	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,150	2,200	2,292	2,467	2,678	2,824	3,131	3,100
15	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,210	2,294	2,590	2,708	2,869	3,010	.....
16	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,733	2,786	2,733	2,850	.....
17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,250	.....	3,030	3,100	.....
Average vital capacity	612	650	755	851	1,022	1,118	1,238	1,412	1,555	1,698	1,870	2,005	2,211	2,449	2,661	2,855	3,017	3,100

TABLE 7.—Normal Boys. Number of Cases, Sitting Height, Average Vital Capacity, Vital Capacity as Determined by Graphic Interpolation Absolute and Percentage Increase in Vital Capacity Accompanying Each 2 Cm. Growth in Stem Length, Vital Capacity as Computed from Sitting Height by Formula, with Formula Used in Computing the Vital Capacity, Corrected Sitting Height and Values Derived from Formula Square of Sitting Height in Cm., Probable Error, Standard Deviation, and Coefficient of Variation

Number of Cases	Sitting Height, Cm.	Average Vital Capacity, C.c.	Mean Vital Capacity as Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity with Each 2 Cm. Growth in Standing Height, Cm.	Percentage Yearly Increase in V. C. with Each 2 Cm. Growth in Standing Height, Cm.	Vital Capacity as Computed by Formula $y = (ax) \pm b$	Formula Used for Each Sitting Height Group $y = (ax) \pm b$	Corrected Sitting Height	S. H.^{2} / V. C.	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
9	53	975	975	...	...	975	y = V. C.	51.4	2.71	....	...	....
7	55	1,036	1,034	59	6.1	1,058	a = Sit. Ht. in Cm.	53.3	2.75	....	...	....
27	57	1,071	1,123	89	8.6	1,141	x = 41.5	55.3	2.72	13.9	151	14.1
51	59	1,190	1,214	91	8.1	1,224	b = −1,224	57.2	2.70	11.3	139	11.7
75	61	1,302	1,307	93	7.7	1,307		59.2	2.68	10.6	187	14.4
78	63	1,425	1,434	127	9.7	1,434	y = V. C.	61.1	2.60	10.9	209	14.7
76	65	1,557	1,576	142	9.9	1,586	a = Sit. Ht. in Cm.	63.1	2.53	12.1	221	14.2
84	67	1,719	1,733	157	10.0	1,788	x = 76	65.0	2.44	12.6	219	12.8
73	69	1,923	1,892	159	9.2	1,890	b = −3,354	66.9	2.42	15.2	217	11 3
78	71	2,054	2,038	146	7.7	2,042		68.9	2.31	15.7	214	10.9
79	73	2,159	2,178	140	6.8	2,194		70.8	2.30	16.4	258	12.0
113	75	2,300	2,326	148	6.8	2,346		72.8	2.28	15.1	239	10.4
85	77	2,497	2,497	171	7.4	2,498		74.7	2.23	18.5	276	11.1
60	79	2,671	2,686	189	7.6	2,686	y = V. C.	76.6	2.18	23.3	287	10.7
91	81	2,881	2,887	201	7.5	2,908	a = Sit. Ht. in Cm.	78.6	2.14	24.5	346	12.0
64	83	3,104	3,104	217	7.5	3,130	x = 111	80.5	2.09	26.2	383	12.3
49	85	3,292	3,342	238	7.7	3,352	b = −6,083	82.5	2.07	31.7	362	12.4
47	87	3,607	3,580	238	7.1	3,574		84.4	1.93	35.5	317	8.8
32	89	3,897	3,796	216	6.0	3,796		86.3	1.96	46.4	328	11.4
16	91	3,913	3,967	171	4.5	3,967	y = V. C.	88.3	1.97			
6	93	4,142	4,123	156	3.9	4,119	a = Sit. Ht. in Cm.	90.2	1.97			
4	95	4,275	4,275	152	3.7	4,273	x = 76.5 b = −2,994	92.2	1.99			

TABLE 8.—Normal Girls. Number of Cases, Sitting Height, Average Vital Capacity, Vital Capacity as Determined by Graphic Interpolation, Absolute and Percentage Increase in Vital Capacity Accompanying Each 2 Cm. Growth in Stem Length, Vital Capacity as Computed from Sitting Height by Formula, with Formula Used in Computing the Vital Capacity, Corrected Sitting Height and Values Derived from Formula, Square of Sitting Height in Cm., Probable Error, Standard Deviation, and Coefficient Variation

Number of Cases	Sitting Height, Cm.	Average Vital Capacity, C.c.	Mean Vital Capacity as Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity with Each 2 Cm. Growth in Standing Height, Cm.	Percentage Yearly Increase in V. C. with Each 2 Cm. Growth in Standing Height, Cm.	Vital Capacity as Computed by Formula $y = (ax) \pm b$	Formula Used for Each Sitting Height Group $y = (ax) \pm b$	Corrected Sitting Height	$\frac{S.H.^2}{V.C.}$	Probable Error of Random Sampling, C.c.	Standard Deviation, C.c.	Coefficient of Variation, per Cent.
1	51	750	750	.....	...	750	(48.6 × Ht.) — 1,728 = V. C.	49.5	3.27			
2	53	1,060	860	+110	14.7	847		51.4	3.08			
10	55	940	956	+ 96	11.2	944		53.3	3.02			
31	57	998	1,038	+ 82	8.6	1.042		55.3	2.95	21.5	177	17.7
48	59	1,107	1,128	+ 90	8.7	1,139		57.2	2.90	12.7	131	11.8
44	61	1,205	1,236	+108	9.6	1,236		59.2	2.84	13.7	159	13.2
71	63	1,386	1,360	+124	10.0	1,360	(65.1 × Ht.) — 2,741 = V. C.	61.1	2.75	10.7	133	9.6
78	65	1,469	1,492	+132	9.7	1,490		63.1	2.67	12.1	159	10.8
69	67	1,636	1,621	+129	8 6	1,620		65.0	2.61	17.3	213	13.0
74	69	1,759	1,747	+126	7.8	1,751		66.9	2.56	16.1	205	11.7
67	71	1,881	1,867	+120	6.9	1,881		68.9	2.54	18.2	221	11.7
57	73	1,952	1,991	+124	6.6	2.011		71.8	2.52	15.6	175	9.0
77	75	2,113	2,124	+133	6.6	2,141		72.8	2.49	17.1	224	10.6
71	77	2,256	2,271	+147	6.9	2,279	(70.5 × Ht.) — 3,149 = V. C.	74.7	2.46	20.5	256	11.3
107	79	2,456	2,420	+149	6.6	2,420		76.6	2.43	20 8	317	12.9
91	81	2,537	2,568	+148	6.1	2,561		78.6	2.41	18.5	261	10.3
89	83	2,740	2,709	+149	5.8	2,702		80.5	2.39	22.1	309	11.3
71	85	2,847	2,843	+134	4.9	2,843		82.5	2.43	22.4	282	9.9
30	87	2,967	2,956	+113	3.9	2,956	(36 × Ht.) + 176 = V. C.	84.4	2.41	44.3	359	12.1
11	89	3,141	3,040	+ 94	3.2	3,028		86.3	2.45			
1	91	3,100	3,100	+ 60	2.0	3,100		88.3	2.51			

sitting height. For both sexes, however, the total relative increase in vital capacity exceeds that for the stem length. Dreyer claims that the vital capacity is related to the square of the sitting height. Whether or not this applies to the period of childhood will be considered later.

The curves of vital capacity as plotted against sitting height are somewhat irregular when drawn from the actual averages. In order to eliminate the minor fluctuations they have been smoothed by means of graphic interpolation (Fig. 3). The means for the vital capacity derived in this manner for each sitting height are recorded in Tables 7 and 8.

In general, the smoothed curves plotted against stem length for each sex are quite similar to those plotted against either age or standing height, showing at first a phase of fairly uniform increase in vital

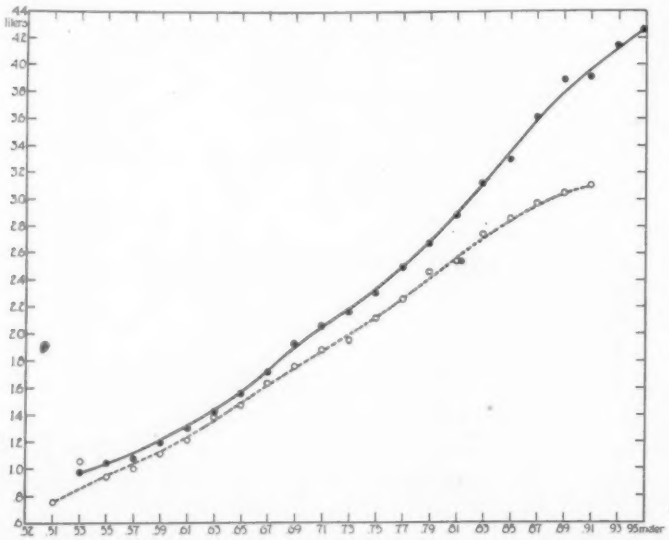

Fig. 3.—Showing smoothed curves representing the average absolute increase in the vital capacity of the lungs as determined by graphic interpolation, for boys with an increase in sitting height from 0.53 to 0.95 meters inclusive and for girls with an increase in sitting height from 0.51 to 0.91 meters inclusive. The actual observed averages at each stem length are respresented by dots for the boys and by circles for the girls. Vital capacity in liters is represented on the ordinate, and sitting height in meters on the abscissa.

capacity, followed by one of more rapid increase, and finally enters a phase of greater retardation in rate of growth of lung capacity. On the whole, the curves tend to assume a concave form throughout the greater extent of their course, the concavity being more marked with the boys than with the girls. At corresponding sitting height the curves show clearly a greater vital capacity for the boys than for the girls, the difference tending to increase as the growth in stem length occurs.

The absolute increase in vital capacity which accompanies each 2 cm. increase in sitting height (Tables 7 and 8) is fairly uniform for each sex at stem lengths ranging from 63 to 75 cm. At this point the absolute increase tends to rise reaching a maximum of 238 c.c. for the boys and 149 c.c. for the girls, at stem lengths of 85 and 79 cm., respectively. With each further increase of 2 cm. in sitting height the gain in vital capacity for each sex progressively diminishes.

The relative increase per 2 cm. growth in sitting height, in general, appears to diminish at first as the stem length increases, reaching a minimum of 6.8 and 6.6 per cent. for the boys and girls, respectively, at 73 cm. sitting height. Subsequently, the relative rate of increase in vital capacity rises slightly with the boys to about 7.7 per cent. when the average sitting height is 85 cm., but later declines again. With the girls the tendency for the relative increase in lung capacity to rise after an earlier period of decline is much less pronounced. It appears, however, that the rapidity of the decline in rate of percentage gain in vital capacity is greatly retarded at sitting heights ranging from 73 to 81 cm.

The relation between sitting height and vital capacity has been fully discussed by Dreyer.[20] This investigator states that the relationship between these two measurements may be expressed correctly by the formula $\frac{SH^n}{V.C.} = K$, where SH = stem length in centimeters, V.C. = vital capacity in cubic centimeters, the power n is approximately 2, and K is a constant. In other words, the vital capacity is related to the square of the sitting height. Dreyer bases this opinion on an analysis of the data of Hutchinson and Schuster, on sixteen personal observations, which to a large extent included observations mainly on adults. The average value for the constant obtained by Dreyer when derived in the above manner is 1.9 for males.

Dreyer measured the stem length by a special technic, seating the subject on the floor with his back against a perpendicular triangular projection, to which a scale is attached. On account of the influence of the muscles, the stem length taken when the subject is seated on a chair is found to be about 3 per cent. greater than those obtained by the method Dreyer used. Since the sitting heights for the children reported in this paper were obtained with the subjects seated on a chair, against a perpendicular wall, it is necessary to reduce my averages 3 per cent. to render them directly comparable with the measurements by Dreyer. The corrected sitting heights are recorded in Tables 7 and 8.

The values for K with the boys (Table 7) as derived from the averages of vital capacity for each corrected sitting height during childhood are by no means a constant. The results obtained show a gradual decrease from an average of 2.71 to 1.93 with an increase in sitting height from 53 to 87 cm. With further growth in stem length

the value determined for K remains practically constant, possibly tending to increase slightly as sitting height increases. It is evident, therefore, that the formula $\frac{SH^3}{V.C.} = K$ is not a correct expression of the relation between the vital capacity and sitting height for the boys. Apparently this relation is not fully established until well into the latter half of childhood. At this period my data for children give a constant (1.93) which is very close to that obtained by Dreyer (1.90) for adults.

With the girls accompanying a growth in stem length from 51 to 83 cm., the values derived for K decrease from 3.27 to 2.39, but subsequently remains fairly constant.

It is evident, therefore, that a constant relation between the square of the sitting height and vital capacity also does not exist with the girls in early childhood, and apparently is not established until the girls obtain a sitting height of approximately 79 cm.

The higher values for the constant with the girls, indicates a relatively smaller vital capacity of the lungs for them than for the boys.

The probable error of random sampling (Tables 7 and 8), as determined for the averages based on sitting height, is comparatively small. In general, for each sex, it apparently tends to increase constantly as sitting height and vital capacity increases.

The standard deviations recorded in Tables 7 and 8 resemble those obtained when the cases are grouped on different basis, tending to increase as vital capacity increases, and averaging higher, as a rule, with the boys than with the girls of corresponding stem length. As for relative variability (coefficients of variation; Tables 7 and 8) there is an apparent decrease in its extent with the shorter groups as they increase in stem length, followed by a later period where variability rises to some extent, and, finally, by a period during which it falls again. For the most part, the coefficients range between 10 and 14. In general, then, the degree of variability in relation to the averages as based on stem length is quite similar to that observed with averages based on standing height.

If the observations at each age are distributed in groups differing 2 cm. in stem length, as is done in Tables 9 and 10, some conception of the variation in vital capacity at any age accompanying differences in stem length can be easily detected. The averages in these tables show that the vital capacities at every age are higher, as a rule, for the children with the greater stem length. When we consider, however, the average vital capacity for the children of similar sitting height but varying in age the differences are much less striking and constant.

5. *Vital Capacity and Body Weight.*—To study the relation between vital capacity and body weight, the data are averaged in Tables 11 and 12 in such manner that each group included subjects who varied not more than 5 pounds in body weight (including clothing).

TABLE 9.—Vital Capacity, in Cubic Centimeters, of Normal Boys for Every 2 Cm. Difference in Sitting Height at Each Age, and Average Vital Capacity for Each 2 Cm. Growth in Stem Length, Combining All Ages

Age in Years	Sitting Height																					
	53	55	57	59	61	63	65	67	69	71	73	75	77	79	81	83	85	87	89	91	93	95
5	950	800	910	1,025	1,008	1,150	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
6	.....	1,125	1,061	1,172	1,184	1,329	1,200	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
7	1,000	1,150	1,150	1,176	1,300	1,323	1,389	1,460	2,200	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
8	.....	.....	1,200	1,300	1,294	1,424	1,556	1,632	1,825	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
9	.....	.....	.....	1,400	1,517	1,496	1,602	1,764	1,796	1,995	2,500	2,575	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
10	.....	.....	.....	.....	1,525	1,817	1,679	1,728	1,920	1,988	2,031	2,200	2,650	.....	.....	.....	.....	.....	.....	.....	.....	.....
11	.....	.....	.....	.....	.....	1,500	1,767	1,783	1,979	2,088	2,110	2,177	2,500	2,500	.....	.....	.....	.....	.....	.....	.....	.....
12	.....	.....	.....	.....	.....	.....	1,700	1,850	1,950	2,050	2,144	2,241	2,406	2,650	2,788	2,800	.....	.....	.....	.....	.....	.....
13	.....	.....	.....	.....	.....	.....	.....	1,800	2,125	2,069	2,267	2,310	2,423	2,604	2,814	3,012	3,334	.....	4,000	.....	.....	.....
14	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,158	2,100	2,396	2,512	2,659	2,813	3,023	3,221	3,395	3,850	3,450	.....	4,250
15	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,400	2,311	2,688	2,788	2,926	3,269	3,288	3,666	3,853	3,769	.....	.....
16	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,388	2,450	2,675	3,617	3,186	3,373	3,606	3,907	4,125	4.300	4.300
17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,550	2,900	3,850	4,000	4,167	4,062	4,300
Average	975	1,036	1,071	1,190	1,302	1,425	1,557	1,719	1,923	2,054	2,159	2,300	2,497	2,671	2,881	3.104	3,292	3,607	3,897	3,913	4.142	4,275

TABLE 10.—Vital Capacity, in Cubic Centimeters, of Normal Girls, for Every 2 Cm. Difference in Sitting at Each Age, and Average Vtal Capacity for Each 2 Cm. Growth in Stem Length, Combining All Ages

Age in Years	Sitting Height																				
	51	53	55	57	59	61	63	65	67	69	71	73	75	77	79	81	83	85	87	89	91
5	750	950	867	859	1,067	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
6	.....	1,150	983	1,068	1,102	1,100	1,250	1,266	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
7	.....	.....	900	1,094	1,098	1,158	1,353	1,400	1,325	1,400	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
8	.....	.....	.....	1,000	1,192	1,253	1,415	1,474	1,483	1,687	1,750	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....
9	.....	.....	.....	.....	1,167	1,383	1,425	1,423	1,540	1,738	1,850	2,000	.....	.....	.....	.....	.....	.....	.....	.....	.....
10	.....	.....	.....	.....	900	1,317	1,404	1,505	1,643	1,749	1,879	2,012	2,175	2,300	2,750	.....	.....	.....	.....	.....	.....
11	.....	.....	.....	.....	.....	1,250	1,431	1,612	1,609	1,773	1,862	1,943	1,975	2,050	2,117	.....	.....	.....	.....	.....	.....
12	.....	.....	.....	.....	.....	.....	1,450	1,450	1,869	1,838	1,867	1,921	2,102	2,258	2,283	2,556	2,700	.....	.....	.....	.....
13	.....	.....	.....	.....	.....	.....	.....	1,700	1,600	1,933	2,000	1,933	2,149	2,236	2,437	2,470	2,713	2,967	2,883	.....	.....
14	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,250	2,230	2,314	2,465	2,595	2,716	2,923	2,958	3,225	.....
15	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,000	2,000	2,150	2,240	2,567	2,686	2,820	2,846	3,029	3,093	.....
16	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,762	2,512	2,550	2,888	3,012	.....	.....
17	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2,250	2,700	3,017	.....	.....	3,100
18	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	3,100	.....	.....	.....
Average	750	1,050	940	998	1,107	1,205	1,386	1,469	1,636	1,759	1,881	1,952	2,113	2,256	2,458	2,537	2,740	2,847	2,967	3,141	3,100

For the boys (Table 11) with an increase in gross weight from 43 to 133 pounds the vital capacity increased from 1,157 to 3,635 c.c. Accompanying this increase in weight, amounting to 209 per cent., the vital capacity has increased 214 per cent. With the girls (Table 12) for a corresponding growth in weight, the lung capacity increases from 1,099 to 3,047 c.c., or 177 per cent. Thus, while with the boys the relative increase in vital capacity on the average exceeds that in weight, just the reverse is true for the girls.

TABLE 11.—NORMAL BOYS. NUMBER OF CASES, BODY WEIGHT, VITAL CAPACITY, AVERAGE VITAL CAPACITY, AVERAGE VITAL CAPACITY AS DETERMINED BY GRAPHIC INTERPOLATION. ABSOLUTE AND PERCENTAGE INCREASE IN VITAL CAPACITY, VITAL CAPACITY AS COMPUTED BY FORMULA, WITH FORMULA USED, 0.72 POWER OF WEIGHT IN GRAMS AND VALUES DERIVED FROM FORMULA $\frac{WO.72}{V.C.}$

No. of Cases	Gross Wt. in Lbs.	Average Vital Capacity, C.c.	Mean Vital Capacity as Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity with Each 5 Lbs. Increase in Weight	Percentage Increase, etc., with Each 5 Lbs. Increase in Gross Weight	Vital Capacity as Determined by Empirical Formula $y = ax \pm b$	Empirical Formula $y = (ax) \pm b$	W .72	*W .72 / V. C.
2	33	988	988	.....	....	988	y = V.C. in Cm.	942	0.953
13	38	1.135	1,090	+102	11.0	1,098	x = 22.0	1,069	0.958
75	43	1,157	1,198	+108	9.9	1,208	b = +263	1,163	0.965
88	48	1,275	1,319	+121	9.2	1,319	a = Wt. in Lbs.	1,258	0.946
78	53	1,458	1,458	+139	10.5	1,458	y = V.C.	1,343	0.921
82	58	1,607	1,611	+153	10.5	1,598	a = Wt. in Lbs.	1,433	0.896
79	63	1,780	1,765	+154	9.6	1,739	x = 28.1	1,526	0.866
97	68	1,926	1,910	+155	8.8	1,879	b = +30	1,606	0.857
86	73	2,062	2,046	+136	7.1	2,020		1,681	0.833
74	78	2,160	2,178	+132	6.5	2,160		1,764	0.812
96	83	2.284	2,311	+133	6.1	2,301		1,850	0.804
60	88	2,506	2,443	+132	5.7	2,441		1,931	0.790
51	93	2,542	2,574	+131	5.4	2,574	y = V.C. in C.c.	2,014	0.783
45	98	2,687	2,704	+130	5.1	2,710	a = Wt. in Lbs.	2,091	0.772
63	103	2.867	2,841	+137	5.1	2,845	x = 27.2	2,171	0.763
41	108	2,922	2.987	+146	5.1	2,982	b = +44	2,247	0.754
38	113	3,176	3.142	+155	5.2	3,118		2,332	0.740
36	118	3,318	3,297	+155	4.9	3,254		2,410	0.733
32	125	3.397	3,443	+146	4.4	3,443		2,493	0.724
27	128	3,696	3.565	+122	3.5	3,565	y = V.C. in C.c.	2,503	0.721
17	133	3.635	3.680	+115	3.2	3.690	a = Wt. in Lbs.	2,629	0.714
27	138	3,745	3,794	+114	3.1	3,816	x = 25.1	2,710	0.713
13	143	3,815	3,929	+135	3.3	3,941	b = +343	2,795	0.709
9	148	4,217	4.957	+128	3.3	4,067		2,861	0.704
8	153	4,181	4,181	+110	2.7	4,183		2,932	0.701

* W = 0.72 power of the net weight in grams.

The straightened curves derived for vital capacity when averaged for weight are somewhat different in character than those previously described. With the boys the smoothed curve (Fig. 4) shows much less sinuosity than the curves plotted either against age, standing and sitting height. As for the girls the curve based on weight is definitely convex in character, and thus altogether different from those derived from averages determined on the other measurements. The convexity

apparently due to the increase in vital capacity occurring at a relatively slower rate than increase in weight during later childhood. As in the case of the curves based on age, standing and sitting height, likewise those plotted against weight may be subdivided into different portions in such manner that each conforms closely to a straight line. It follows, therefore, that the vital capacity may be computed from the body weight by means of the same empirical formula used for the other curves.

TABLE 12.—NORMAL GIRLS. NUMBER OF CASES, BODY WEIGHT, VITAL CAPACITY, AVERAGE VITAL CAPACITY, AVERAGE VITAL CAPACITY AS DETERMINED BY GRAPHIC INTERPOLATION. ABSOLUTE AND PERCENTAGE INCREASE IN VITAL CAPACITY, VITAL CAPACITY AS COMPUTED BY FORMULA, WITH FORMULA USED, 0.72 POWER OF WEIGHT IN GRAMS AND VALUES DERIVED FROM FORMULA $\frac{WO.72}{V.C.}$

No. of Cases	Gross Wt. in Lbs. Including Clothing	Average Vital Capacity, C.c.	Average Vital Capacity as Determined by Graphic Interpolation, C.c.	Absolute Increase in Vital Capacity with Increase in Weight	Percentage of Increase in Vital Capacity with Increase in Weight	Vital Capacity as Computed from Formula $y = (ax) \pm b$	Formula $y = (ax) \pm b$	W 0.72	$\frac{*W .72}{V. C.}$
30	38	930	930	.....	....	930	y = V. C.	1,069	1.149
54	43	1,009	1,086	+156	16.8	1,075	a = Wt. in Lbs.	1,163	1.069
63	48	1,244	1,237	+151	13.9	1.220	x = 29	1,263	1.042
86	53	1,388	1,382	+145	11.7	1,365	b = —172	1,356	0 999
76	58	1.527	1.521	+135	9.8	1,510		1,446	0.963
74	63	1,651	1,656	+132	8.7	1,655		1,570	0.948
63	68	1,798	1,788	+129	7.8	1,788	y = V. C.	1,624	0.914
78	73	1,918	1,917	+129	7.2	1.908	a = Wt. in Lbs.	1,713	0.900
60	78	2.029	2,046	+124	6.5	2 028	x = 24	1,800	0 887
61	83	2,207	2,170	+122	6.0	2,148	b = +156	1,885	0 879
69	88	2,271	2,292	+114	5.2	2,268		1,987	0.878
64	93	2,428	2,406	+103	4.5	2,388		2,047	0.860
64	98	2,520	2,509	+ 84	3.5	2,508		2,132	0.850
57	103	2,620	2,593	+ 67	2.7	2,593	y = V. C.	2,208	0.851
48	108	2,720	2.660	+ 68	2.6	2,660	a = Wt. in Lbs.	2,291	0 862
36	113	2,628	2,728	+ 77	2.8	2,727	x = 13.4	2,361	0.867
32	118	2,803	2.805	+ 89	3.3	2,794	b = +1,213	2,527	0.882
28	123	2,900	2.894	+ 78	2.8	2,861		2,589	0.884
15	128	3,037	2.972	+ 55	1.9	2,928		2,663	0.889
15	133	3.047	3,027	+ 34	1.1	2,995		2,743	0.896
14	138	3,061	3,061	+ 34	1.1	3,062			
6	143	2,725							
4	148	3,000							
4	158	3,388							
2	173	2,900							
28	123	2,900	2,894	+ 78	2.8	2,861		2,589	0.884
1	193	2,900							

* W = 0.72 power of the net weight in grams.

The actual values for the constants to be used for different weight groups may be determined by referring to Tables 11 and 12. The vital capacities computed from formula agree fairly closely with the actual averages and also with those as determined by graphic interpolation.

Dreyer [20] claims that the relation between the vital capacity and body weight may be correctly expressed by the formula $\frac{W^a}{V.C.} = K$, where

W is the net weight in grams, V.C. = vital capacity in cubic centimeters, the power n is 0.72, and K = a constant. The average value obtained for K by Dreyer for data collected largely from male adults is 0.69.

In order to compare this relationship to body weight for children with Dreyer's findings for adults it was first necessary to correct my weights for clothing. The net weight for my cases has been determined by computing the weight of the clothing and deducting this from the gross weight. In estimating the weight of clothing the factors published by Bardeen [5] were used and the net weights obtained in this manner are tabulated in Tables 11 and 12.

With the boys the values obtained for K by means of the formula $\frac{W^{n}}{V.C.} = K$ are seen to decrease constantly as growth in body weight

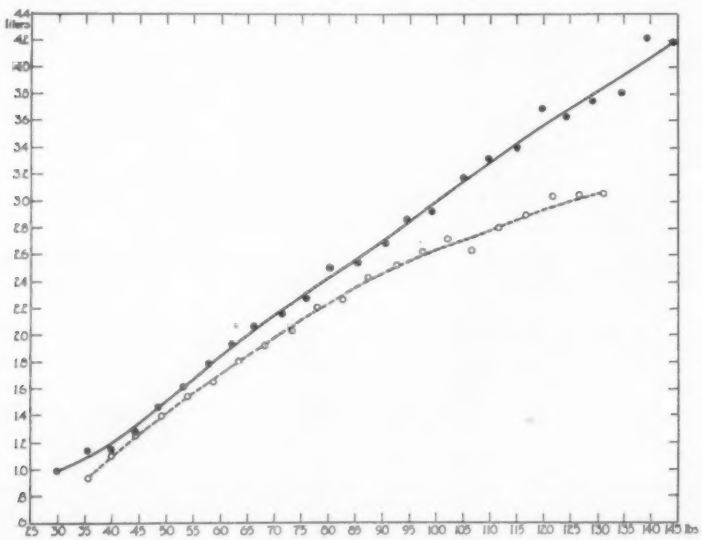

Fig. 4.—Showing smoothed curves representing the average absolute increase in the vital capacity of the lungs as determined by graphic interpolation for boys with an increase in net body weight from 30 to 145 pounds inclusive, and for girls with an increase in body weight from 35 to 130 pounds inclusive. The actual observed averages at each net body weight are represented by dots for the boys, and by circles for the girls. Vital capacity in liters is represented on the ordinate, and net weight in pounds on the abscissa.

occurs, diminishing from an average of 0.955 at a net weight of 40 pounds (18,100 gm.) to 0.701 when weighing 143.8 pounds (65,400 gm.). This latter value, 0.701, is quite similar to that (0.69) obtained by Dreyer for adults. It is evident from these results that the constant relation between the 0.72 power of the weight and vital capacity is not established with boys until the net body weight is in the neighborhood of 140 pounds.

With the girls the value for K with an increase in net weight from 40 to 92 pounds decreases from 1.089 to 0.850. With further increase in weight the value for K seems to increase slightly. For the girls also, then, the formula advocated by Dreyer is not a correct expression of the relation of vital capacity to body weight for children. It is not until the latter part of childhood that the above relation is fully established. At corresponding age, the values obtained for K (Tables 11 and 12) are higher for the girls than for the boys. It follows from this, as shown in numerous other instances, that lung capacity is relatively smaller for girls than for boys of similar size

TABLE 13.—NUMBER OF CASES, AGE, WEIGHT, STANDING HEIGHT, VITAL CAPACITY, NORMAL VITAL CAPACITY EXPECTED FOR CORRESPONDING STANDING HEIGHT, PER CENT. OF DEVIATION FROM NORMAL, AND NOTE REGARDING DYSPNEA FOR BOYS AND GIRLS WITH MITRAL REGURGITATION

Average No. of Cases	Average Age, Years	Average Net Weight, Lbs.	Average Standing Height, Cm.	Average Vital Capacity, C.c.	Average Normal Vital Capacity for Corresponding Height, C.c.	Average per Cent. of Deviation from Normal	Notes Regarding Dyspnea
Boys							
8	11	74.6	142	2,131	2,182	— 2.4	No dyspnea on exertion
Girls							
17	12	77.4	144	1,939	2,005	— 3.2	No dyspnea on exertion
Boys							
1	7	40	112	900	1,160	—22.4	Spleen enlarged; moderate dyspnea; râles in lungs
2	15	57	131	775	1,780	—56.5	Dyspnea pronounced
3	11	55	133	1,600	1,850	—13.5	Dyspnea on mild exertion
4	15	61	148	1,300	2,230	—41.7	Dyspnea on slight exertion
5	16	102	162	1,350	3,180	—57.5	Dyspnoic constantly
6	18	122	174	2,100	3,860	—45.6	Dyspnea on slight exertion
Girls							
1	9	40	121	950	1,360	—30.1	Dyspnoic
2	11	62	129	1,100	1,580	—30.4	Slight dyspnea
3	12	52	127	900	1,525	—41.0	Mild dyspnea on exertion
4	11	62	129	1,100	1,590	—30.8	Moderate dyspnea
5	13	87	145	900	2,110	—57.3	Severe dyspnea
6	13	80	146	1,250	2,150	—41.9	Moderate dyspnea
7	20	117	150	1,500	2,310	—35.1	Moderate dyspnea
8	14	83	152	1,200	2,400	—50.0	Dyspnea on slight exertion
9	16	120	163	1,950	2,830	—31.0	Considerable dyspnea

Although the present investigation is devoted mainly to a consideration of the vital capacity of the lungs of normal and healthy children, a series of observations were made on a considerable number of subjects suffering from pathologic conditions of the heart and lungs. A brief discussion of the results obtained in each disease is given below.

6. *Vital Capacity and Mitral Insufficiency.*—Records were obtained for fourteen boys and twenty-six girls suffering from mitral insufficiency of varying degrees of severity (Table 13). The data for eight boys and seventeen girls show the vital capacity to average practically normal for their standing height in spite of the presence of a

leaking mitral valve. These children, as a group, are able to play and romp almost as vigorously as normal children without suffering from dyspnea to any extent. With the more severe cases, however, the vital capacity is seen to be greatly reduced, the reduction in some cases amounting to more than 50 per cent., and in such instances dyspnea is a very prominent symptom. In general, it may be said, that the tendency to become dyspneic is directly proportional to the degree to which the vital capacity is reduced. Ruebow,[14] Bittorf and Forschbach,[23] Beebak, Bie and Maar,[16] Plesch,[17] Wilson and Edwards,[24] and others have also noted the vital capacity to be reduced by diseases of the heart. Siebeck [15] attributes the decrease in vital capacity in heart disease to a change in the elasticity of the lungs resulting from engorgement of the pulmonary circulation. From a study of 124 adults suffering from various types of heart disease, Peabody and Wentworth [18] have decided that, in general, patients with a vital capacity of 90 per cent. or more of the normal standard adopted for their sex and height have little or no abnormal tendency to dyspnea. If reduced to 70 or 90 per cent. of the normal, dyspnea appears to be an unusual exertion, while if reduced to 40 or 70 per cent. of the normal the activities of these patients must be greatly restricted. Those with a vital capacity of less than 40 per cent. of the normal are usually confined to bed, and the mortality is high with this group.

Repeated observations made of a few children having mitral disease show that the vital capacity increases as the clinical condition improves. The record for one girl with mitral regurgitation and rheumatic fever is given in Figure 5. The record of a girl suffering from cardiac decompensation following a heavy day's work is given in Figure 6. In each instance the vital capacity is seen to progressively increase, and the hospital records show a similar improvement in their condition clinically. With the older girl (Fig. 6) the reduction in vital capacity recorded on the twenty-sixth day of illness followed a tonsillectomy done two days previously. When dismissed from the hospital on the twenty-eighth day of illness the vital capacity recorded was considerably higher even than the normal expected for corresponding standing height.

Peabody and Wentworth and Ulrich and Nathanson [25] also present records for adults that show fluctuations in the vital capacity to correspond with changes in the patient's condition. Periodic measure-

23. Bittorf, A., and Forschbach, J.: Untersuchungen über die Lungenfüllung bei Krankheiten, Ztschr. f. klin. Med. **70,** 1910.

24. Wilson, M. G., and Edwards, D. J.: Standards for Normal Vital Capacity for Children: The Lung Capacity in Certain Intrathoracic Conditions, Am. J. Dis. Child. **22**:443 (Nov.) 1921. Diagnostic Value of Determining Vital Capacity of Lungs of Children, J. A. M. A. **78**:1107 (April 15) 1922.

25. Ulrich, H. L., and Nathanson, M. H.: The Capacity of the Lungs in Cardiac Disease, Minnesota M. **4**:721, 1921.

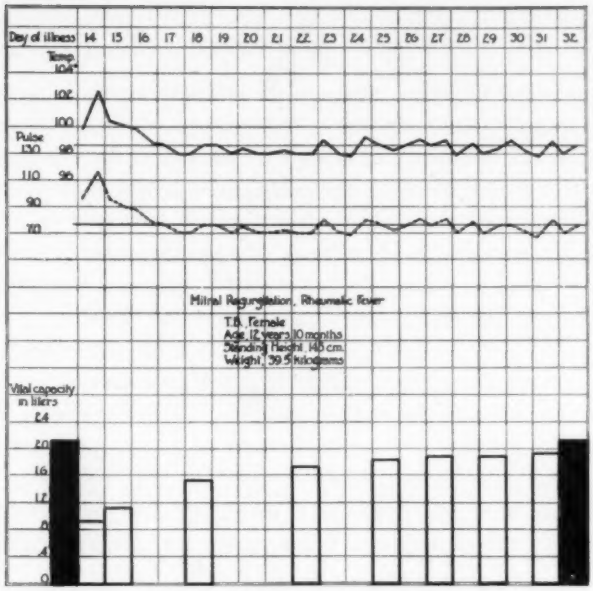

Fig. 5.—Showing the changes in body temperature, pulse rate, and vital capacity of the lungs from the fourteenth to the thirty-first day of illness, for a girl having acute rheumatic fever, and mitral regurgitation. The solid black rectangles represent the normal vital capacity for corresponding standing height; the blank rectangles represent the observed vital capacity on successive days.

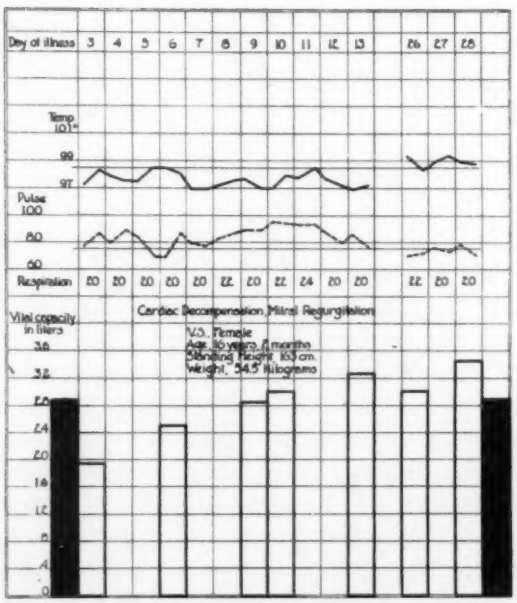

Fig. 6.—Showing the changes in body temperature, pulse rate, rate of respiration and vital capacity of the lungs from the third to the thirteenth and for the twenty-sixth to the twenty-eighth day of illness, for a girls cardiac decompensation, and mitral regurgitation. The solid black rectangles represent the normal vital capacity for corresponding standing height; the blank rectangles represent the observed vital capacity on successive days.

ment of the vital capacity, therefore, affords a simple and practical method of detecting fluctuations in the general condition of the patient.

This study of a limited number of children suffering from disease of the mitral valve shows that in milder cases in which the vital capacity is practically normal very little respiratory discomfort is experienced even on severe exertion. In more severe cases, however, the tendency to develop dyspnea in a measure parallels the reduction in vital capacity.

7. Vital Capacity and Acute Bronchitis.—In Table 14 are recorded the average vital capacities for seventeen boys and fourteen girls, respectively, having acute bronchitis, which shows that the vital capacity is considerably reduced by this condition. The individual data show the degree of reduction to be rather marked in the more severe cases. Often the presence of considerable mucus in the trachea and bronchi provokes peroxysms of coughing toward the end of expiration, and thus prevent its completion. Even with the patients showing the

TABLE 14.—NUMBER OF CASES, AGE, WEIGHT, STANDING HEIGHT, VITAL CAPACITY, NORMAL VITAL CAPACITY FOR CORRESPONDING STANDING HEIGHT, AND PER CENT. OF DEVIATION FROM NORMAL FOR BOYS AND GIRLS HAVING ACUTE BRONCHITIS

Average Number of Cases	Average Age, Years	Average Gross Weight, Lbs.	Average Standing Height, Cm.	Average Vital Capacity, C.c.	Average Normal Vital Capacity for Corresponding Standing Height	Average Per Cent. Deviation from Normal
Boys..... 17	10	65.3	131	1,450	1,795	—18.9
Girls..... 14	10	63.1	131	1,354	1,650	—17.9

greatest reduction in vital capacity due to their bronchitis, there was little complaint of dyspnea. Peabody and Wentworth also record that the vital capacity of the lungs may be considerably reduced following the inception of acute bronchitis.

8. Vital Capacity and Labor Pneumonia.—During the past year vital capacity records were obtained of eleven children during an attack of pneumonia. The data for nine of the children (Table 15) show the vital capacity to be enormously reduced early in the course of the disease. The degree of reduction is greatly out of proportion to the extent of the pneumonic process even with patients not suffering from pleurisy. For example, one boy (Case 1) on the fifth day of illness, although free from pleurisy, was able to expire only 450 c.c., which is about one third the normal expected for corresponding standing height. At the same time, fluroscopic examination demonstrated the pneumonia to be limited to the lower lobe of the right lung. In this instance, although, roughly, one fourth or less of the total lung tissue was involved by the pneumonic process, nevertheless, the vital capacity

was reduced by three fourths. In other cases the reduction is equally striking. The general weakness of the patient is evidently a factor in reducing the vital capacity.

The initial reduction in vital capacity, and its gradual return to normal following recovery from lobar pneumonia is clearly shown for two boys in Figures 7 and 8. In the case of the older patient (Fig. 7), on the second day of illness the vital capacity was reduced 35 per cent. At this time the clinical diagnosis was somewhat uncertain. On the fourth day, however, a diagnosis of lobar pneumonia was definitely established, and the vital capacity was now reduced approximately 71 per cent. Following the crisis the vital capacity gradually

TABLE 15.—NUMBER OF CASES, AGE, STANDING HEIGHT, WEIGHT, INITIAL VITAL CAPACITY, DAY OF ILLNESS, VITAL CAPACITY AT EACH SUCCEEDING READING, NORMAL VITAL CAPACITY FOR CORRESPONDING STANDING HEIGHT, PER CENT. OF DEVIATION FROM THE NORMAL FOR BOYS AND GIRLS WITH LOBAR PNEUMONIA

No.	Standing Height, Cm.	Net Weight, Lbs.	Initial Vital Capacity and Day of Illness	Vital Capacity at Each Succeeding Reading						Normal Vital Capacity for Corresponding Standing Height	Per Cent. of Deviation from the Normal
Boys			4	5	6*	9	12				
1	118	50	350	450	800	1,050	1,250	.....	.....	1,340	— 6.7
			6	8*	14						
2	116	51	750	1,000	1,225	.....	.....	.....	.....	1,280	— 4.3
			3	4	5	7*	8	9	10		
			4	7*	8	9	14	50	71		
3	134	65	400	600	700	900	750	800	1,200	1,880	—36.2
			3	13	20	25					
4	135	59	850	1,350	1,600	1,700	.....	.....	.....	1,920	—11.5
Girls			1	6	7*	10	12				
5	106	39	150	750	775	800	850	.....	.....	950	—10.6
			7*	9	12	14	15	16			
6	123	...	700	750	800	900	1,000	1,100	.....	1,410	—22.0
			4	6*	8	10					
7	43	...	350	1,000	1,100	1,250	.....	.....	.....	2,030	—38.4
			10	12	15	19					
8	133	62	800	900	1,300	1,550	.....	.....	.....	1,510	+ 2.6
			6	9	12	16	20	23	26		
9	155	960	300	675	1,000	1,500	1,650	2,000	2,050	2,520	—18.7

* Day of crisis.

increased and by the seventeenth day of illness was only slightly more than 17 per cent. below the normal expected for corresponding standing height. For this patient a record of vital capacity was obtained nine months following his recovery from pneumonia. During this interval the records show an increase in vital capacity from 2,850 c.c. to 3,500 c.c. This indicates that following recovery from an attack of pneumonia, subsequent growth in lung capacity may proceed in a rather satisfactory manner.

One child (Case 3) failed to recover promptly. Areas of consolidation persisted in one lung for a long time which, finally, terminated in an empyema. The records show that during the period studied the

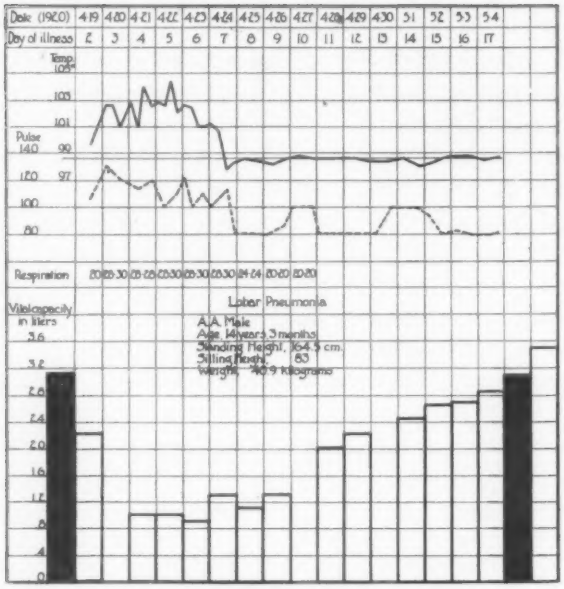

Fig. 7.—Showing the changes in body temperature, pulse rate, rate of respiration and the vital capacity of the lungs from the second to the seventeenth days of illness, for a boy having lobar pneumonia. The solid black rectangles represent the normal vital capacity for corresponding standing height; the blank rectangles represent the observed vital capacity on successive days.

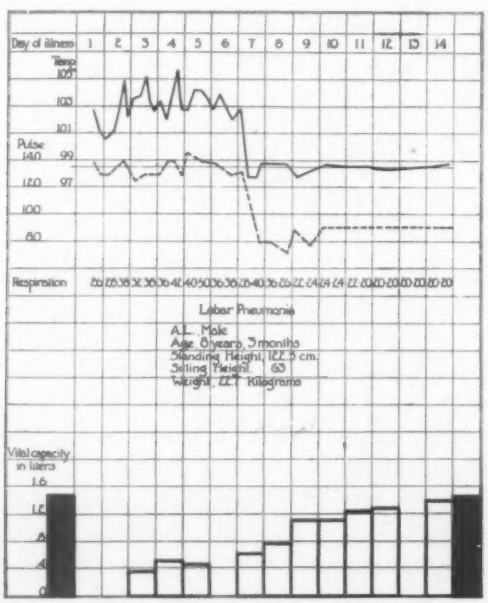

Fig. 8.—Showing the changes in body temperature, pulse rate, rate of respiration, and the vital capacity of the lungs from the first to the fourteenth day of illness, for a body having lobar pneumonia. The solid black rectangles represent the normal vital capacity for corresponding standing height; the blank rectangles represent the observed vital capacity on successive day.

vital capacity remained considerably below normal. Accompanying slight improvements in the patient's general condition, the vital capacity increased slightly, but decreased again when he felt worse. At the time the last observation was made the vital capacity was still 36 per cent. below the expected normal.

During the course of this investigation the disappearance of physical signs was observed in several instances to precede the complete return of vital capacity to normal.

9. *Vital Capacity and Bronchial Asthma.*—Several patients subject to attacks of asthma visited the dispensary during the past winter. The

TABLE 16.—NUMBER OF CASES, AGE, WEIGHT, STANDING HEIGHT, VITAL CAPACITY, NORMAL VITAL CAPACITY FOR CORRESPONDING STANDING HEIGHT, PER CENT. OF DEVIATION FROM NORMAL AND CLINICAL NOTES FOR BOYS AND GIRLS SUBJECT TO BRONCHIAL ASTHMA

No. of Cases	Age, Years	Gross Weight, Lbs.	Standing Height, Cm.	Vital Capacity, C.c.	Normal Vital Capacity for Corresponding Standing Height	Per Cent. of Deviation from Normal	Clinical Notes: Duration of Disease
Boys							
1	6	32	109	750	1,080	—30.6	Many râles present
2	6	39	113	775	1,190	—34.9	Many râles present
3	6	45	118	1,600	1,340	+19.4	No râles present
4	6	41	118	1,550	1,340	+15.7	No râles present
5	8	48	123	1,900	1,500	+26.7	No râles present; asthma for 6 years
6	7	48	123	1,450	1,500	— 3.3	Few râles present; asthma for 4 years
7	8	57	124	800	1,530	—47.7	Many râles present
8	10	64	132	1,900	1,810	+ 5.0	Asthma for 3 years; no râles present
9	11	64	132	1,800	1,810	— 0.5	
10	12	68	140	2,350	2,100	+11.9	Few râles present; asthma for 4 years
11	13	68	145	2,350	2,300	+ 2.2	Asthma for 9 years
12	11	72	146	1,950	2,340	—16.7	Asthma for 2 years; no râles present
13	14	104	155	3,200	2,760	+15.9	Eight years
Girls							
1	7	38	119	900	1,300	—30.8	Many râles present; 3 years
2	9	46	120	1,275	1,330	— 4.1	No râles present
3	9	50	130	1,800	1,620	+11.1	Few râles present
4	13	65	...	2,500	.....	.....	No râles present; 7 years

vital capacity records for these children are tabulated in Table 16. The data show that the vital capacity, even with children who have experienced severe asthmatic attacks for several years, may be practically normal during intervals when they are free from symptoms. However, immediately following acute attacks, when the lungs show marked evidence of the disease, the vital capacity may be reduced greatly (Cases 1, 2, 7 and 14). The greatest reduction noted in this study amounted to 47.7 per cent. in Case 7. For one patient (Case 2) the vital capacity was recorded during an acute attack, and also again at a later time when he was free from symptoms. In this instance,

during the attack, his vital capacity was nearly 35 per cent. below normal, whereas after the symptoms disappeared it actually exceeded the normal expected for his standing height almost 12 per cent.

From this study of a limited number of cases the conclusion seems justified that while bronchial asthma may cause an enormous reduction in the vital capacity immediately following an acute attack, nevertheless, the reduction promptly disappears and the vital capacity returns to normal as the symptoms subside. Even in cases subject to the disease for several years there is apparently no permanent reduction in vital capacity.

Peabody and Wentworth note that with adults the vital capacity becomes greatly lowered during asthmatic attacks. Four patients tested soon after acute attacks were found to have vital capacities of 56, 60, 85 and 102 per cent. of the normal, respectively. Seven observations on six patients made when they were in their best condition between

TABLE 17.—AGE, NUMBER OF CASES, NET WEIGHT, STANDING HEIGHT, VITAL CAPACITY, NORMAL VITAL CAPACITY FOR CORRESPONDING STANDING HEIGHT, AND PER CENT. THAT OBSERVED VITAL CAPACITY DEVIATED FROM THE NORMAL, FOR CHILDREN OF FRESH AIR SCHOOL

Average Age, Years	Average Number of Cases	Average Weight, Kg.	Average Standing Height, Cm.	Average Vital Capacity, C.c.	Average Normal Vital Capacity for Corresponding Standing Height, C.c.	Average per Cent. Deviation from Normal
Boys..... 11	46	33.0	142.8	2,055	2,182	—5.8
Girls..... 11	51	32.3	140.0	1,842	1,925	—4.3

asthmatic spells showed a range in vital capacity between 65 and 122 per cent. of the normal. In only one case was it below 85 per cent.

10. *Vital Capacity and Tuberculosis.*—In Table 17 are recorded the vital capacities of forty-six boys and fifty-one girls, all of whom were attending what is known as the fresh air school. Children are eligible for admission to this school only in case they have a positive Pirquet (tuberculin) reaction, and show some clinical evidence of tuberculosis, such as slight daily elevation in temperature, frequent attacks of bronchitis or failure to gain in weight properly. Physical examination of the lungs in most instances was practically negative, except for the usual presence of a positive D'Espine sign, and, as a rule, the roentgen-ray findings were limited to enlarged peribronchial lymph glands at the hilus of the lungs. The condition of this group of cases is probably fairly representative of the usual early type of quiescent tuberculosis encountered in children.

The vital capacity of the lungs for the boys (Table 17), while tending to be slightly low in many cases, in general, agrees fairly well

with normal averages for corresponding height. The reading for only five of the boys appeared to be definitely subnormal. However, on comparing the stem length of each of these boys with the normal as found by Boas [26] for corresponding height, in each instance the observed sitting height is found to be considerably less than that expected of boys of their height. Since standing height seemed rather abnormal with these boys as compared with their sitting height, it was thought that the records would probably agree more closely with averages of vital capacity based on stem length. When the observed vital capacities was compared with the normal expected for corresponding stem length in each instance, the vital capacity appears to be practically normal as compared with the normal for corresponding sitting height. In general, then, it is doubtful if the lung capacity of any of these forty-six boys can be considered as definitely abnormal in this respect.

For the girls (Table 17) the average vital capacity agrees fairly well with the normal expected for corresponding standing height. In only four instances were the readings significantly subnormal.

While the number of observations is too small to permit of very sweeping conclusions, nevertheless, the data indicate that the vital capacity is usually normal in children reacting positively to the intracutaneous application of old tuberculin and showing little additional evidence of tuberculosis aside from a positive D'Espine and a comparatively poor health record. Certain of these children, however, have an abnormally low vital capacity. These may be the ones who are most likely to develop serious forms of the disease in the future. Later determinations of their vital capacity, if showing a further decrease, would probably give immediate clue to any change in their pulmonary condition.

In addition to these children showing a minimum evidence of tuberculosis, the lung capacity also of twenty-six boys and seventeen girls with definite tuberculosis of bone was studied. The data obtained from this series of patients are recorded in Table 18. The average value for the readings for each sex appear to be practically normal for standing height. For a few of the individuals, however, the lung capacity was abnormally low, especially with the more serious cases. For one boy the reduction amounted to 40 per cent. This boy has a marked kyphosis of the midthoracic region which is completely ankylosed. The marked deformity and resultant mechanical interference with inspiration probably accounts partly for his low vital capacity, especially since the lungs are apparently normal.

For another patient the vital capacity was 46.4 per cent. below the normal expected. This patient was in a very poor general condition,

26. Boas, F., and Wissler, C.: Statistics of Growth, U. S. Education Commision **1**, 1904.

weighing only 49 pounds, and has an active tuberculosis of the lower lumbar spine with a psoas abscess and several discharging sinuses. The roentgen ray also revealed a marked shadow about the hilus and considerable mottling throughout the lung field. Six months later the vital capacity was recorded a second time and during this period was found to have decreased from 1,050 c.c. to 750 c.c., a decrease of about 29 per cent. At this time his condition was definitely worse than at the previous examination. In this instance one, no doubt, would

TABLE 18.—NUMBER OF CASES, AGE, WEIGHT, STANDING HEIGHT, VITAL CAPACITY, NORMAL VITAL CAPACITY FOR CORRESPONDING STANDING HEIGHT, LOCATION AND EXTENT OF DISEASE FOR BOYS HAVING TUBERCULOSIS OF BONE AND LUNGS

Number of Cases	Average Age, Years	Average Net Weight, Lbs.	Average Standing Height, Cm.	Average Vital Capacity, C.c.	Average Normal Vital Capacity for Corre-Standing Height	Average per Cent. of Deviation from Normal	Average Location and Extent of Tuberculosis
Girls							
17	10	50.5	125	1,401	1,470	— 4.9	Tuberculosis of hip, knee, spine, etc.
Pulmonary Tuberculosis							
1	13	55	135	575	1,770	—67.5	Advanced pulmonary; extensive cavity formation
2	16	82	157	1,000	2,610	—61.7	Advanced pulmonary; extensive cavity formation
3	18	110	151	1,000	2,350	—57.4	Advanced pulmonary; extensive cavity formation
Boys							
26	11	59.5	128	1,542	1,700	— 9.3	Tuberculosis of hip, knee, spine, etc.
Pulmonary Tuberculosis							
1	9	61	123	1,000	1,490	—32.9	Lungs, with effusion (incipient)
2	11	85	145	2,250	2,300	— 2.2	Lungs (incipient)
3	12	60	145	1,000	2,300	—56.5	Lungs (chronic fibroid), peritoneum
4	15	107	177	2,900	3,980	—27.1	Lungs (moderately advanced)
5	16	103	157	2,700	2,880	— 6.2	Lungs (incipient)
6	18	50	*	750	4,300*	—82.6	Spine, lungs
7	17	81	157	1,100	2,880	—61.8	Lungs (advanced), spine

* V. C. for corresponding age.

suspect the above to be true merely from considering the vital capacity record alone, even without knowing other facts about the case.

For the seventeen girls (Table 18) having tuberculosis of the bone the average for the vital capacity is practically normal. The volumes for two subjects of this group, however, were abnormally low, being 25.6 and 35.5 per cent., respectively, below the normal for corresponding height. With these two girls observations six months later show in each instance an increase of about 100 c.c., in vital capacity. The gain in lung capacity registered by these patients during this period

probably indicates a better prognosis than in the case mentioned above in which the vital capacity decreased to a considerable extent.

In only a few instances was it possible to obtain observations of the vital capacity of the lungs of children with definite and active pulmonary tuberculosis. Data for ten such cases are recorded in Table 18. Of the incipient cases only one boy (Case 1) has a vital capacity significantly subnormal. In this instance the presence of a pleural effusion probably accounts for a considerable portion of this reduction. In Case 2 the lung capacity is practically normal. The roentgenogram of this patient's lungs revealed a rather large shadow extending from the hilus out into the parenchyma of the right lung; and the Pirquet reaction was strongly positive. About one months after the vital capacity was recorded for the first time, this boy had a tonsillectomy following which he developed a tuberculous meningitis and promptly died. This case illustrates, therefore, that a normal vital capacity may be obtained even in the presence of a pathological pulmonary condition of the most serious potentialities.

Among the advanced cases, two patients (Cases 3 and 9) have died during the past few months. In these cases, however, death was not unexpected for in both the tuberculous process was very extensive. It is interesting to note that of the advanced cases four patients (Cases 6, 7, 8 and 9) were ambulatory and suffered no dyspnea on mild exertion in spite of the fact that their vital capacities were reduced to about one third their normal value. This observation stands out in marked contrast to these symptoms noted in cardiac disease for with a similar reduction in vital capacity dependent on some pathologic condition of the heart, dyspnea is a constant and very prominent symptom.

Many investigators, including Hutchinson, Wintrich, Scheevoogt, Arnold, Fabius, Schönfeld,[27] Siebeck, Wilson and Edwards, and others, have observed that the vital capacity may be reduced greatly in the presence of pulmonary tuberculosis. Recently Dreyer and Burrell[20] have been able to classify cases of pulmonary tuberculosis as normal or as incipient, moderate or advanced, without seeing the patients, knowing only their vital capacity and various body measurements.

From a study of patients with incipient tuberculosis Garvin, Lundsgaard and Van Slyke[28] found the vital capacity to be reduced as a result of an increased residual air. In moderately advanced and advanced cases the vital capacity was substantially reduced principally

27. Schonfeld, L.: Ein Beitrag zur Lehre von der Spirometrie, Diss. Berlin 1882.

28. Garvin, A.; Lundsgaard, C.; Van Slyke, D. D.: Studies of Lung Volume II Tuberculous Men, J. Exper. M. **27**:87, 1918. Studies of Lung Volume III, Tuberculos Women, J. Exper. M. **27**:129, 1918.

as a result of diminished total lung capacity. The residual air was normal, as a rule, although in a few cases an increase in residual air also contributed to the decrease in vital capacity.

Wittich, Meyers and Jennings [29] found the vital capacity to decrease relative to body weight, in direct proportion to the amount of disease present.

CONCLUSIONS

From this study it appears evident that the determination of the vital capacity of patients having diseases of the heart and lungs is of distinct clinical value. Although single observations are of importance, repeated measurements are of much greater value, for changes in the pathologic condition of these organs is immediately reflected by fluctuations in the vital capacity. It is undoubtedly inadvisable to use the determination of the vital capacity to the exclusion of other means of making a diagnosis, for occasionally this measurement is very misleading and might easily lead to serious error if relied on alone. However, with the exercise of proper judgment and care, one usually is able to use the information gained through the measurement of the vital capacity safely and profitably in determining not only the presence but also roughly the extent and progress of the disease of the heart and lungs.

SUMMARY

The more important results of the present investigation may be summarized briefly as follows:

1. At either corresponding age, standing height, sitting height, or body weight the vital capacity of the lungs is constantly greater with the boys than with the girls, the difference increasing with the especial rapidity during the latter half of childhood.[30]

2. The curves of vital capacity as plotted against age, standing and sitting height, in general, pursue a sinuous course, showing a fairly uniform increase at first, followed later by a period of more rapid growth in lung capacity, which later diminishes in rate. The curve plotted against body weight for the boys shows a more uniform course, while that for the girls has a definitely convex form.

3. The absolute annual increase in vital capacity, in general, is fairly uniform during early childhood, increases in rapidity about the time of puberty, and later declines.

4. The relative increase in vital capacity apparently decreases at first, becomes relatively more rapid about the time of puberty, and

29. Wittich, F. W., Myers, J. A., and Jennings, F. L.: A Study of the Effect of Pulmonary Tuberculosis on Vital Capacity, J. A. M. A. **75**:1249 (Nov. 6) 1920.

30. Emerson, P. W., and Green, H.: Vital Capacity of the Lungs of Children, Am. J. Dis. Child. **22**:202 (Aug.) 1921.

then proceeds at a slower rate again. The period of acceleration in the rate of growth in lung capacity begins and terminates at an earlier age with the girls than with the boys.

5. Throughout childhood the vital capacity formed a greater proportion of the estimated adult maximum (3,200 c.c.) for women with the girls, than with the boys, the adult maximum of the latter be estimated as approximately 4,300 c.c. In general, then, with respect to lung capacity, at corresponding ages the girls may be considered relatively more mature than the boys.

6. The vital capacity of the lungs apparently reaches a maximum near the twentieth year with the boys, and probably slightly before this time with the girls.

7. On using the proper values for the constant, the vital capacity may be computed [31] fairly accurately from the age, the standing height, the sitting height or the body weight by means of the empirical formula $y = (AX) \pm b$, where y = vital capacity in cubic centimeters; a = (either age in years, standing height or sitting height in cubic centimeters or body weight in pounds), X = a constant, and $\pm b$ = a second constant. The proper values for the constants may be determined by referring to the appropriate tables.

8. The probable error of random sampling increases as the vital capacity increases, but in all instances is relatively small, amounting to less than 25 c.c., as a rule. Therefore, averages obtained from an infinitely large number of comparable series would vary only slightly from those obtained in the present limited series of observations.

9. The standard deviation (absolute variability), as a rule, increases as the vital capacity increases, and usually is greater with the boys than with the girls of corresponding age and size.

10. The coefficient of variation (relative variability) for each sex decreases during early childhood, increases about the period of puberty and later declines again. The extent of the dispersal of the individual observations from the averages, appears to be smaller when averaged in groups differing 5 cm. in standing height or 2 cm. in stem length, than when averaged according to age in years, as estimated from the nearest birth day.

11. The constant relation between the vital capacity and the square of the sitting height, and also between the 0.72 power of the body weight in grams and the vital capacity apparently is not established until the latter part of childhood.

12. Children may have a normal vital capacity even in the presence of a definite insufficiency of the mitral valves, and these individuals show

31. West, H. F.: Clinical Studies on the Respiration. VI. A Comparison of Various Standards for the Normal Vital Capacity of the Lungs, Arch. Int. Med. **25**:306 (March) 1920.

little abnormal tendency to dyspnea on exertion. In more serious cases the vital capacity is greatly reduced, and the tendency to respiratory embarrassment is proportional to the reduction in lung capacity.[32]

13. When the condition of the heart improves, the vital capacity gradually increases.

14. Acute bronchitis produces a temporary reduction in vital capacity, which in severe cases may be very marked.

15. During the course of lobar pneumonia the vital capacity rapidly undergoes an enormous reduction, even in cases free from pleurisy. The reduction is altogether out of proportion to the extent of the pneumonic process in the lungs. Following the crisis, the vital capacity gradually returns to normal, in a comparatively short time. Physical signs of the disease, however, may disappear before the normal vital capacity is reached. In complicated cases the return of vital capacity to normal may be greatly delayed.

16. While bronchial asthma may cause an enormous reduction in the vital capacity immediately following an acute attack, nevertheless the reduction promptly disappears as the symptoms subside. Even in children subject to the disease for several years, there is apparently no permanent reduction in the vital capacity of the lungs.

17. With children showing slight evidence of tuberculosis, such as a positive Pirquet (tuberculin) reaction, and having a poor health record, and also with those having definite tuberculosis of bone, the vital capacity is normal, as a rule. In a few instances, however, the lung capacity is sufficiently subnormal to lead one to suspect some pulmonary involvement.

In incipient pulmonary tuberculosis the vital capacity may be practically normal, even in the presence of a very active and virulent infection in the lungs.

In advanced pulmonary tuberculosis the vital capacity of the lungs is greatly reduced, in proportion to the extent of the disease process.

18. The abnormal tendency to dyspnea on exertion is much more marked when the reduced vital capacity is caused by disease of the heart, than when caused by disease of the lungs alone, even in instances where the reductions are similar in extent.

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32. Pearce, R. G.: The Cardiorespiratory Mechanism in Health and Disease, Arch. Int. Med. **27**:139 (Feb.) 1921.

DEHYDRATION IN NEW-BORNS I*

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Since water is the medium in which the physical and chemical processes of the body take place, variations in the amount of the body fluids must be of great importance in the changes which occur during health and disease. The demonstration by Balcar and his associates [1] that fever may be produced by dehydrating animals makes the subject of particular interest in new-borns in whom fever and dehydration occur so frequently.

A number of observers believe that transient fever in the new-born is due to dehydration. This belief is based on the fact that the fever usually occurs at the time of the greatest weight loss in infants who have lost much weight. The loss of weight, it is assumed, indicates a water loss.

Objection has been raised to this conception of "inanition fever" [2] because many new-born babies lose considerable amounts of weight without developing fever while others develop fever after only slight losses of weight. In a subsequent paper we shall attempt to show that in the large majority of new-born babies with fever the plasma water is diminished. In this paper I propose to discuss the relation of weight loss to dehydration in new-born babies and to show the fallacy of drawing conclusions from the weight changes alone regarding the water content of the new-born.

The ease with which various tissues take up and lose water varies considerably. The muscles and skin apparently are very susceptible to changes in the water content of the organism. Tobler [3] dehydrated animals so that they lost from 25 to 30 per cent. of their body weight in a few days. He found that most of the water loss was accounted for by the loss in water from the muscles and skin. The blood and soft tissues lost comparatively little fluid. A number of observers [4]

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1. Balcar, J. D.; Sansum, W. D., and Woodyatt, R. T.: Arch. Int. Med. **24**:116 (Aug.) 1919.

2. Grulee, C. G., and Bonar, B. E.: Am. J. Dis. Child. **22**:44 (July) 1921.

3. Tobler, L.: Arch. f. exper. Path. u. Pharmakol. **62**:431, 1909.

4. Engels: Arch. f. exper. Path. u. Pharmakol. **51**:346, 1904. Bogert, L. J.; Underhill, F. P., and Mendel, L. B.: Am. J. Physiol. **41**:189, 219, 229, 1916. Smith, A. H., and Mendel, L. B.: Am. J. Physiol. **53**:323, 1920.

have also shown that when saline is injected into the vein of an animal, the fluid quickly leaves the blood going for the most part to the muscles and skin thus increasing their water content. Recently Weed and McKibben [5] found that the water content of the brain is readily varied.

For a long time it was thought that, despite variations in the water content of the tissues, the blood water remained constant. It was believed that the tissues acted as a reservoir from which water might be withdrawn and to which water might be added without significantly altering the concentration of the blood. It is now known, however, that the blood water may change under a number of conditions.[6] Rogers, in 1909,[7] using the hematocrit, found that the blood may lose more than 60 per cent. of its water in cholera. Recently Underhill and his coworkers [8] showed that, accompanying the pulmonary edema seen in war gas poisoning and influenza, the blood may become concentrated. Numerous observations also show that in acute intestinal intoxication in infants the blood may become markedly concentrated.

When an organism loses water it does not lose it uniformly from all the organs. Certain organs (muscle, skin, brain) apparently give up water very readily, while others, such as the blood and soft tissues, remain comparatively unchanged until the water loss becomes marked. In some instances, however, fairly wide fluctuations may occur in the plasma water without corresponding changes in the water content of the baby.[9]

Fairly accurate methods are available for determining changes in the water percentage of the blood and plasma. Variations in the red cell content of the blood (also hemoglobin, dry residue of the blood, corpuscular volume, etc.) indicate changes in the percentage of water of whole blood. Variations in the plasma protein per cent. indicate changes in the plasma water per cent. The total plasma volume may also be determined. Since the red cell content of the blood during the first two weeks of life varies rapidly, due to causes other than changes in the blood water, determinations of the red cells, hemoglomin, etc., are of little value as indices of changes in the blood water content.

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5. Weed, F. S., and McKibben, L. H.: Am. J. Physiol. **48**:531, 1919.

6. Cushing, H., and Foley, F. E. B.: Proc. Soc. Exper. Biol. & Med. **17**: 1580, 1920.

7. Rogers, L.: Philippine J. Sc. **4**:99, 1909.

8. Underhill, F. P.: Arch. Int. Med. **23**:753 (June) 1919. Underhill, F. P., and Ringer, M.: J. A. M. A. **75**:1531 (Dec. 4) 1920.

9. For example, in a baby weighing 3,600 gm., 100 c.c. of saline by mouth reduced the serum protein per cent. from 7.55 to 5.93, indicating an increase of 27 per cent. in the plasma water. The amount of fluid given was only 3 per cent. of the body weight. In order to produce a corresponding increase in the water content of the rest of the tissues it would have been necessary to give nearly a liter of fluid.

In our experiments the refractive index of serum was used to measure changes in plasma water. Since, under most conditions, the plasma proteins are fairly constant, any change in their concentration is taken to indicate a change in the plasma water. The consistent results obtained in these experiments seem to indicate that in the newborn baby this assumption is, in the main, justified.

Method.—The serum protein percentage was determined with the Abbe refractometer. Distilled water was read with each series of readings. The factor for the nonproteins, determined in older infants [10] by the method of Robertson [11] was used. The calculation of the serum protein per cent. was made according to Reiss.[12]

The serum protein percentage is of importance not only because it measures changes in plasma water but also because it gives a measure of the osmotic resistance to the passage of water from the blood. The capillary wall acts as a semipermeable membrane [13] through which water and crystalloids (salts, sugar, urea, etc.) may pass freely but through which colloids are unable to pass. (In special situations, notably the liver, the plasma proteins are able to pass through the capillary wall, but in the main, Bayliss believes, the capillaries are impermeable to colloids). These colloids, the plasma proteins mainly, exert an osmotic pull on water which, according to Starling [14] amounts to from 30 to 35 mm. of mercury.

In the capillary we have, therefore, a force, the blood pressure, tending to squeeze water out of the circulation, while opposing it is the osmotic pressure of the plasma proteins which tends to hold water in the capillaries. One can readily understand that an increase in the plasma protein concentration, whether due to a diminution in the plasma water or to an actual increase in the plasma proteins, results in an increase in the resistance to the passage of water from the capillaries.

It is possible that the refractive index of the serum of the newborn baby gives only a rough estimate of the protein concentration of plasma. Lewis and Wells [15] have shown that in the serum of newborns euglobulin is almost entirely absent. The use of the factor 0.00172 (Reiss) for the calculation of the serum proteins is based on the assumption that the serum globulin and serum albumin exist in the same proportion as in the serum of adults. However, though the absolute values may not be correct, the relative differences of con-

10. Bakwin, H.: Am. J. Physiol. **60**:343, 1922.
11. Robertson, T. B.: J. Biol. Chem. **22**:233, 1915.
12. Reiss, E.: Ergebn. d. inn. Med. u. Kinderh. **10**:531, 1913.
13. Bayliss, W.: J. Pharmacol. & Exper. Therap. **15**:29, 1920.
14. Starling: J. Physiol. **19**:312, 1896.
15. Lewis, J., and Wells, H. G.: J. A. M. A. **78**:863 (March 25) 1922.

centration are sufficiently large so that the significance of the results is in no way diminished.

For purposes of comparison a chart showing the serum protein concentration of babies during the first week of life is shown(Fig. 1). Only afebrile babies are represented. It will be seen that the serum protein per cent. is usually between 6 and 7. It rises only rarely above 7.5, and in but three instances did it rise above 8. After the fourth day it is unusual to find the serum proteins above 7 per cent.

Hemoglobin was determined by the Palmer method.[16] The carbon monoxid standard was checked repeatedly with the Van Slyke and Stadie method[17] over a period of several months and found to check within 2 per cent. The hemoglobin results are charted in grams per hundred cubic centimeters of blood.

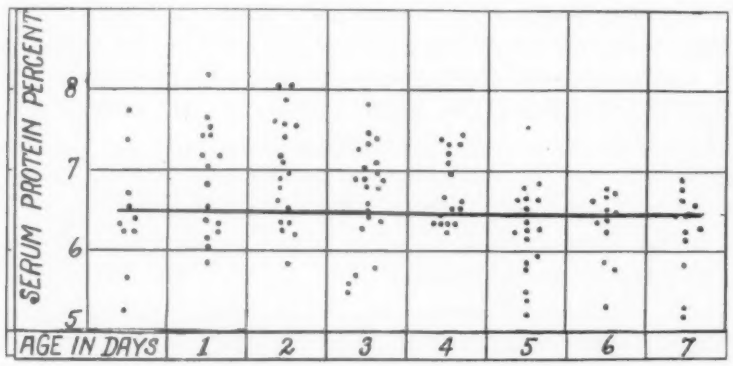

Fig. 1.—The serum protein per cent. of babies during the first week of life. The dots represent the individual determinations, the heavy black line the average serum protein per cent. Most of the points lie between 6 and 7 per cent., rarely above 7.5 per cent.

A loss of skin elasticity indicates that the water content of the skin is diminished below normal. In this discussion the degree of inelasticity has been designated by the number of pluses, 1 plus indicating a slight but distinct diminution in skin elasticity, 2 plus a more marked degree of inelasticity, etc.

THE RELATION BETWEEN CHANGES IN WEIGHT AND CHANGES IN THE WATER CONTENT OF THE NEW-BORN BABY

There are no direct methods for measuring variations in the water content of the tissues during life. The only indications of such changes are variations in the body weight and the condition of the skin. Freund[18] showed that the daily up and down curve of an infant's

16. Palmer, W. W.: J. Biol. Chem. **33**:119, 1918.
17. Van Slyke, D. D., and Stadie, W. J.: J. Biol. Chem. **49**:1, 1921.
18. Freund, W.: Jahrb. f. Kinderh. **59**:421, 1904.

weight is largely accounted for by changes in the water content of the organism. In the new-born a number of factors must be considered in estimating the significance of weight changes. The meconium, urine in the bladder, regurgitated allantoic fluid and vernix caseosa account for some of the weight loss. A part of it is also due to an actual loss of body tissue or stored food reserve during the starvation. This loss, according to Benedict and Talbot [19] amounts to about 17 gm. of fat per day for a baby weighing 3.76 kg. (from 35 to 40 gm. of protein or carbohydrate). The rest of the weight loss is due to water. For example, if a baby loses 300 gm. during the first three days of life, a not usually large loss, about 100 gm. are due to the loss of weight from meconium, etc.,[20] about 115 gm. at most, are due to loss of tissue

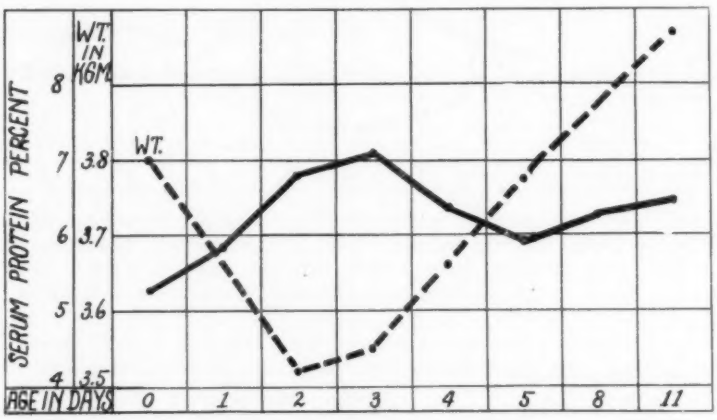

Fig. 2.—Case 1. Increase in serum protein per cent. accompanying the loss of weight after birth. The serum proteins at birth were 5.23 per cent. Accompanying the loss of 280 gm. the serum proteins rose on the third day to 7.09. With the increase in weight between the third and fourth days the serum proteins fell to normal and remained there during the rest of the period of observation.

or food reserve and the remaining 85 gm. are due to water loss. Since most infants get some food during this time from the breast, and, at the New York Nursery and Child's Hospital, from whey which is given after each breast feeding, the water loss is probably considerably greater than the rough calculations just given indicate. It seems reasonable to assume, therefore, that the weight loss gives a rough index of the water loss in a new-born baby.

Variations in the Water Content of Babies at Birth.—In estimating the significance of weight changes in regard to the water content of the new-born baby at any particular time, it is essential that the water

19. Benedict, F., and Talbot, F.: Carnegie Institution, Publication No. 233, 1915, p.11.

20. Vierordt-Gerhardt: Handbuch für Kinderkrankheiten **1**:118, 1877; quoted by Griffith: Diseases of Children, Philadelphia **1**:46, 1921.

content of the baby at birth be considered. For example, a baby who has a large water reserve at birth can lose a great deal more weight without becoming dehydrated than can another infant who is dehydrated at birth.

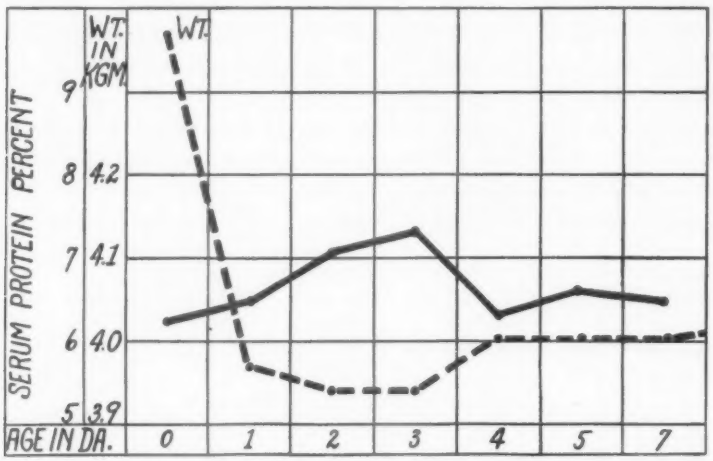

Fig. 3.—Case 2. Increase in the serum proteins accompanying the loss of weight after birth. In this baby the loss of 430 gm. was accompanied by a rise of the serum proteins from 6.28 to 7.31 per cent. With the increase in weight the serum protein concentration dropped to normal and remained there.

TABLE 1.—DEHYDRATION ACCOMPANYING THE LOSS OF WEIGHT AFTER BIRTH

Case	Age, Days	Weight, Gm.	Serum Protein, %	Hemoglobin, Gm. per 100 C.c.	Skin
1	Birth	3,800	5.23	....	Normal
(Fig. 2)	1	.....	5.87	....	1 plus, inelastic
	2	3,520	6.80	....	1 plus, inelastic
	3	3,520	7.09	....	1 plus, inelastic
	4	3,660	6.34	....	Normal
	5	3,750	5.96	....	Normal
	8	.....	6.28	....	Normal
	11	3,970	6.45	....	Normal
2	Birth	4,370	6.28	18.3	Normal
(Fig. 3)	1	3,970	6.51	17.8	1 plus, inelastic
	2	3,940	7.09	19.2	1 plus, inelastic
	3	3,940	7.31	17.2	1 plus, inelastic; Temp. in a.m. 38 C.
	4	4.040	6.34		
	5	4,010	6.62	20.3	Normal
	7	4,040	6.45	....	Normal

The case reports show that the water content of the baby at birth may vary widely. Usually, as the baby loses weight during the first few days after birth, the skin becomes inelastic and the plasma concentration increases indicating a diminution in skin and plasma water (Table 1).

Two babies (Cases 1 and 2) showed no evidence of dehydration at birth. The skin turgor was good and the plasma concentration

(serum protein per cent.) not increased. During the first three days of life they lost considerable amounts of weight. The weight loss was accompanied by an increase in the plasma concentration and a loss of skin elasticity (diminution of plasma and skin water). As the babies started to gain the blood and skin returned to normal.

An increase in the serum protein concentration during the loss of weight after birth has been noted by Rott [21] and by Rusz.[22] The hemoglobin per cent. in Case 2 varied from day to day without relation to weight or serum protein per cent.

There are babies, however, who apparently are born with a large water reserve. In these babies the weight loss is unaccompanied by any detectable evidence of dehydration (Table 2). Such cases have been described by Utheim.[23] Two babies (Cases 3 and 4) lost considerable amounts of weight without showing any evidence of skin or

TABLE 2.—Loss of Weight Unaccompanied by Dehydration

Case	Age, Days	Weight, Gm.	Serum Protein, %	Hemoglobin, Gm. per 100 C.c.	Skin
3	Birth	3,150	6.57	22.3	Normal
	1	2,980	6.39	19.8	Normal
	2	2,840	6.28	20.5	Normal
	3	2,810	5.75	....	Edema of dorsum of hand and feet
	4	2,810	....	19.9	Normal
	5	2,770	5.40	19.2	Normal
	6	2,840	5.87	....	Normal
4	Birth	3,840			
	1	3,780	6.34	....	Normal
	2	3,580	6.68	....	Normal
	3	3,580	6.51	....	Normal
	5	3,600	5.64	....	Normal
	7	3,600	6.16	....	Normal
	11	3,090	6.11	....	Normal

plasma dehydration. It is possible that the water reserve was large at birth, or that the water lost came from the deeper tissues such as the muscles, etc., where it could not be detected by the methods employed. It is possible that the weight loss was accompanied by a destruction of the plasma proteins so that a diminution in the plasma water might have occurred which was compensated for by a loss of plasma protein. The fact that the skin as well as the plasma did not become dehydrated, despite the weight loss, seems to indicate that the water reserve in these babies was large at the start. Whatever the explanation for the failure of the plasma to become concentrated, however, the fact remains that, in spite of the loss of considerable amounts of weight in certain babies, the serum protein concentration remains normal. This is of considerable importance in view of the close rela-

21. Rott, F.: Ztschr. f. Kinderh. (Originalien) **1**:43, 1910.
22. Rusz, E.: Monatschr. f. Kinderh. (Originalien) **10**:360, 1912.
23. Utheim, K.: Saertryk av Norsk Mag. f. Lægevidensk., 1921, p. 104.

tionship between fever in the new-born and an increase in the serum concentration.

In the two cases just described the weight loss was unaccompanied by any evidence of dehydration, probably because the babies had large water reserves at birth. There are babies, however, who are dehydrated at birth. In one of the babies referred to the skin alone was involved; in

TABLE 3.—SKIN DEHYDRATION AT BIRTH

Case	Age, Days	Weight, Gm.	Serum Protein, %	Hemoglobin, Gm. per 100 C.c.	Skin
5	Birth	3,750			
	1	3,750	6.16	19.2	2 plus, inelastic, dry
	2	3,490	7.14	22.1	2 plus, inelastic, dry
	3	.....	....	20.7	2 plus, inelastic, dry
	4	3,440	7.31	20.3	2 plus, inelastic, dry
	5	3,490	6.63	21.0	2 plus, inelastic, dry
	6	3,550	6.28	21.3	2 plus, inelastic, dry
	8	3,580	6.86	19.6	Doubtful
	12	3,600	6.57	....	Normal

the two others, the skin and plasma. Case 5 was first observed fifteen hours after birth (first day). At this time the skin was dry and inelastic, indicating a diminution in skin water. The plasma water was normal. As the baby lost weight the plasma quickly became concentrated. With the increase in weight on the fifth day the plasma returned to normal. The skin, however, remained dehydrated for several days afterward. Case 6 was observed three hours after birth;

TABLE 4.—SKIN AND PLASMA DEHYDRATION AT BIRTH

Case	Age, Days	Weight, Gm.	Serum Protein, %	Skin
6	Birth	2,950	7.43	3 plus, inelastic, dry
	1	2,890	7.61	3 plus, inelastic, dry
	2	.....	8.02	3 plus, inelastic, dry
	3	2,890	6.91	3 plus, inelastic, dry
	4	3,010	6.97	3 plus, inelastic, dry
	6	3,130	6.61	Normal
	12	3,290	6.51	Normal
7	Birth	3,320		
	1	3,290	7.48	3 plus, inelastic, dry
	2	3,290	8.06	2 plus, inelastic, dry
	3	3,140	7.48	2 plus, inelastic, dry
	4	3,140	7.20	3 plus, inelastic, dry
	6	3,260	7.55	Dry, no loss of elasticity
	8	3,320	6.74	Normal

another baby (Case 7) was seen thirteen hours after birth. During this time the latter had lost 30 gm., an insignificant loss in weight and hardly sufficient to account for the dehydration. At the first observation both showed distinct evidence of skin and plasma dehydration as indicated by the marked skin inelasticity and the high serum protein concentration. At this time they were more dehydrated than most of the afebrile babies who had lost large amounts of weight.

The desiccation persisted as long as the babies continued to lose weight. In Case 6 the serum protein concentration returned to normal two days before the skin. In Case 7 the skin regained its elasticity before the plasma water had returned to normal.

At birth, apparently, the water content of the baby may show many degrees of variation. At one extreme is the watery baby who may lose considerable amounts of weight without showing any evidence of dehydration. At the other extreme is the dried out, desiccated baby who at birth shows a dry, inelastic skin and a concentrated plasma. Between these two extremes all gradations exist.

Increase in the Plasma Water Without Increase in Weight.—Another point to be considered in estimating the significance of weight changes in regard to the water content of the baby is the transportation

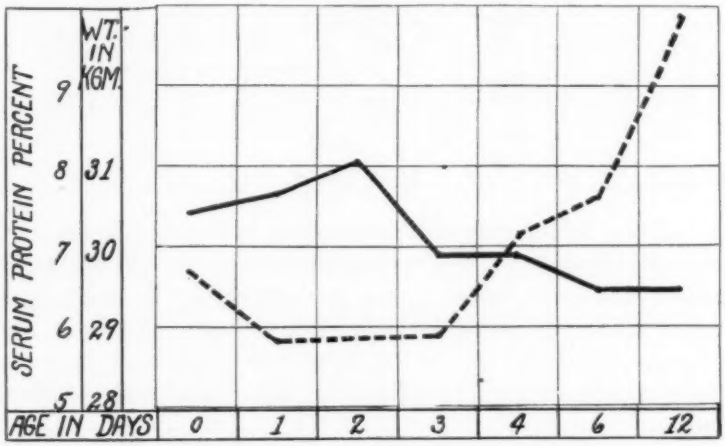

Fig. 4.—Case 6. Dehydration at birth. On the birthday the serum protein per cent. was 7.43 per cent., indicating a concentrated plasma. A loss of a few grams was accompanied by a rise of the serum proteins to 8.02 per cent., a high figure rarely reached by afebrile babies. Between the third and fourth days the serum proteins fell to 6.91 and remained normal thereafter.

of water in the baby from one tissue to another. In a few instances we have noted a fall in the serum protein concentration from one day to another without any increase in weight.

In two babies (Cases 8 and 9) the plasma became more watery despite the fact that the weight did not increase. The decrease in the plasma concentration may have been due to an increase in plasma water or to a destruction of plasma proteins. Whatever the explanation, however, the fact remains that an increase in the plasma water per cent. may occur without any increase in weight.

The Relation Between Skin and Plasma Water in the New-Born.—When dehydration develops in a new-born baby it is usual for the skin

to lose its elasticity before the plasma has become concentrated and to persist for some time after the plasma water has returned to normal (Cases 1, 2, 5 and 6). It is not at all unusual to find the skin inelastic in a baby whose plasma is normal or even dilute (Case 1, second day). Marked degrees of skin inelasticity are usually accompanied by an increase in the plasma concentration although this is by no means always the rule (Case 6).

TABLE 5.—INCREASE IN PLASMA WATER WITHOUT CHANGE OF WEIGHT

Case	Age, Days	Weight, Gm.	Serum Protein, %	Hemoglobin, Gm. per 100 C.c.	Skin
8	Birth	3,260			
	1				
	2	3,180			
	3	3,120	....	7.3	1 plus, inelastic
	4	3,120	8.49	15.9	1 plus, inelastic
	5	3,120	6.91		
	6	3,120	6.57	15.6	Normal
	7	3,120	6.57	....	Normal
9	Birth	3,690			
	1	3,690	8.49	....	1 plus, inelastic, dry
	2	3,630	7.31	....	2 plus, inelastic, dry
	3	3,630			
	4	3,690			
	5	3,750			

An increase in the plasma concentration is usually accompanied by a loss of skin elasticity. In a few instances the skin was normal while the plasma was concentrated (Case 7, sixth day). In one baby with a serum protein per cent. of 9.40, the skin, although very loose, showed no evidence of dehydration. In the fever cases, particularly, the plasma may be markedly dehydrated with only slight impairment of the skin elasticity.

The Relation Between Hemoglobin Per Cent. and the Water Content of the New-Born Baby.—In a number of cases, daily determinations of the hemoglobin were made. No relation, however, was found between the weight changes, serum protein percentage and the hemoglobin.

SUMMARY AND CONCLUSIONS

1. Dehydration was studied in new-born babies by correlating weight changes, skin elasticity and serum protein concentration (plasma water).

2. Uusually a marked weight loss in the new-born baby is accompanied by a loss of skin elasticity and an increase in the serum concentration (indicating a diminution in skin and plasma water).

3. There are babies, however, who, despite relatively large weight losses, show no evidence of dehydration of the skin or plasma In these babies, presumably, the water reserve of the tissues at birth is large.

4. On the other hand, there are babies who show evidence of dehydration at birth. The skin alone may be desiccated (loss of skin elasticity without increase in the serum protein concentration) or both skin and plasma may be involved (loss of skin elasticity and increase in serum protein concentration).

5. Since the water content of the baby varies so widely at birth it is impossible to tell from the weight curve alone whether or not a baby is dehydrated.

6. The plasma water per cent. may increase even though there is no increase in weight.

7. Impairment of skin elasticity may, and often does, occur without increase in the plasma concentration. Increase in the plasma concentration is usually, but not always, accompanied by an inelastic skin.

It is with pleasure that I acknowledge my indebtedness to Dr. H. M. Painter and Dr. Fellows Davis, Jr., for the use of material from the Obstetrical Service of the New York Nursery and Child's Hospital. I wish to thank Dr. J. F. Sammis and Dr. L. C. Schroeder for their stimulating interest in the work. Thanks are due to Dr. Donald McGill, Resident, for his cooperation and to Miss Ethel Viele for her valuable assistance.

DEHYDRATION FEVER IN NEW-BORNS II*

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The frequent occurrence of a transient fever during the first few days of life is now generally recognized. The exact nature of the fever has not yet been satisfactorily explained. Holt,[1] who gave the first adequate description of the condition in 1895 ascribed it to starvation and called it inanition fever.

Of the many hypotheses which have been advanced to explain this peculiar fever the one which has received the most experimental support is that which attempts to explain the fever as due to water loss. The observation originally made by Holt that the fever usually occurs at the time when the weight loss is greatest and disappears when the baby starts to gain has been amply confirmed.[2] Holt also showed that the fever can be made to disappear by administering fluids.[3] He mentions instances of fever in older infants on low fluid intakes which cleared up when fluid was given. Stewart[4] succeded in reducing the incidence of fever in new-borns by giving lactose solutions between nursings. Faber[5] practically prevented fever in new-borns by complemental feedings of whole milk dilutions.

In 1910, Erich Mueller[6] described two cases of fever in older infants who were receiving very little fluid. In both instances the temperature fell to normal when fluids were given. Grulee and Bonar[7] described similar instances of fever in infants who were receiving concentrated diets. In these babies, as in Mueller's, the fever disappeared when fluids were given. In one of their babies the red cell content of the blood was high during the fever falling after the temperature had become normal. They interpreted the high red cell count as indicating a diminution in the blood water. Marriott[8] has suggested that the fever which occurs so frequently in babies with severe diarrhea may be due to dehydration.

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1. Holt, L. E.: Arch. Pediat. **12**:560, 1895.
2. Von Reuss, A. R.: Ztschr. f. Kinderh. (Originalien) **4**:32, 1912. Heller, F.: Ztschr. f. Kinderh. (Originalien) **4**:55, 1912.
3. See also Peteri, I.: Jahrb. f. Kinderh. **80**:612, 1914.
4. Stewart, C. A.: J. A. M. A. **78**:1865 (June 17) 1922.
5. Faber, H. K.: Am. J. Dis. Child. **24**:56 (July) 1922.
6. Mueller, E.: Berl. klin. Wchnschr. **47**:673, 1910.
7. Grulee, C. G., and Bonar, B. E.: Am. J. Dis. Child. **21**:220 (March) 1921.
8. Marriott, W. M.: Harvey Lectures, 1920.

In 1915, Balcar, Sansum and Woodyatt [9] produced marked degrees of fever in animals by severely dehydrating them with intravenous injections of hypertonic glucose solutions given rapidly. They suggested that the fever was due to a failure of normal heat loss because of lack of water evaporation from the body.

Barbour and Howard [10] showed that the fall in temperature following the administration of glucose to dogs with fever is accompanied by a hemodilution (decrease in the hemoglobin per cent. of the blood). They also found that in colon bacillus fever in animals and in human febrile cases antipyrin, quinin and salicylates reduce the blood concentration. Schmitt and Sansum [11] succeeded in reducing the temperature of patients with pneumonia and influenza several degrees and in some instances below 37.8 C. (100 F.) by giving large quantities of fluid by mouth. In a recent review Barbour [12] strongly favors the view that "body temperature is regulated by water shifting under nervous control."

The investigations described in this paper were undertaken in an attempt to determine the relationship between dehydration and fever in the new-born. Three methods were used for estimating dehydration, the plasma concentration (refractive index of serum), the condition of the skin and the weight loss.

The applicability of these methods to a study of dehydration in the new-born has been discussed in the previous paper.[13] It was pointed out that the water content of babies at birth varies widely. Usually a distinct weight loss during the first days after birth is accompanied by a loss of skin elasticity and an increase in the plasma concentration indicating a loss of water from the skin and plasma. In some babies, however, comparatively large weight losses occur without the development of skin or plasma dehydration. In these babies, presumably, the water reserve at birth is large. On the other hand, some babies at birth show evidence of skin and plasma dehydration (loss of skin elasticity and increase in serum protein concentration). From the weight curve alone, therefore, one cannot draw conclusions as to the water content of the new-born baby; the water content of the baby at birth must also be considered. This variation in the water content of the baby at birth explains the occurrence of dehydration fever in new-borns without very large weight losses (dehydration at birth).

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9. Balcar, J. O.; Sansum, W. D., and Woodyatt, R. T.: Arch. Int. Med. **24**:116 (July) 1919.
10. Barbour, H. G., and Howard, A. J.: Proc. Soc. Exper. Biol. and Med. **17**:150, 1920.
11. Schmitt, E. C., and Sansum, W. D.: Cal. State J. Med. **19**:371, 1921.
12. Barbour, H. G.: Physiol. Rev. **1**:295, 1921.
13. Bakwin, H.: Am. J. Dis. Child. **24**: (Dec.) 1922.

It also explains the lack of fever in certain babies who lose a great deal of weight (large water content at birth).

Procedure and Methods.—Daily determinations were made on a group of normal and febrile babies. Blood for refractive index (and in a number of cases for hemoglobin) was drawn from the toe without pressure from three to four hours after a feeding. The temperature of the baby was taken at the time of the puncture. Details of the technic for the serum protein (refractive index of serum) and hemoglobin determinations are given elsewhere.[13]

The only study recorded in the literature on the plasma water in new-born babies with fever is that of Utheim.[14] She concluded that fever in the new-born may occur without a simultaneous increase in the concentration of the plasma. The refractometric method was used.

According to Reiss [15] in older infants with fever due to various infections, the plasma is more dilute than normal (refractometric determination of serum). A large number of determinations made in this laboratory during the past three years showed no increase in the serum protein concentration during the fever associated with various infections. We have had little opportunity to study the plasma concentration in new-borns with fever due to infections. In one baby 5 days old with sepsis and a temperature of 40.6 C. (105 F.) the serum protein was low—5.87 per cent. In another new-born baby with diarrhea and a temperature of 38.3 C. (101 F.) the serum protein was 6 per cent., which is a low normal.

Observations were made on fifty new-borns with fever. Of these, one case each of sepsis, diarrhea and hemorrhagic disease have not been used in the preparation of Figure 1. In these cases the serum protein concentration was not increased. The remaining forty-seven cases with which this paper concerns itself conformed fairly closely to the classical description of inanition fever. The fever usually occurred from forty-eight to ninety-six hours after birth in babies who had lost considerable amounts of weight. In some of the babies, however, the onset of the fever was within the first thirty-six hours after birth and in some the weight loss was not marked.

The skin usually showed some evidence of dehydration (dry, inelastic skin). In most instances this was not more marked than in the afebrile babies. The association between fever in new-borns and a loss of skin elasticity is not a close one. Some of the babies, particularly those with a high serum concentrations, appeared to be quite ill. They were prostrated, apathetic, limp and at times looked pale. Other babies, however, with equally high serum concentrations did not appear ill.

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14. Utheim, K.: Saertryk av Norsk. Mag. f. Laegev., 1921, p. 104.
15. Reiss, E.: Ergebn. d. inn. Med. u. Kinderh. **10**:531, 1913.

In Figure 1 are shown the results of serum protein determinations on normal and febrile babies. The dots represent the afebrile babies, the crosses the febrile. The lower line represents the average normal serum protein percentages each day; the upper line the average for the febrile babies. The serum protein per cent. of normal new-borns lies fairly constantly between 6 and 7. In only three of the babies did it rise above 8 per cent. In the febrile babies the serum protein was usually above 7.5 per cent. In five babies it rose above 9 per cent., as high a figure as is seen in many severe cases of acute intestinal intoxication in older infants. These results show that in the group of cases of fever studied the plasma concentration was definitely increased.

In eight babies the serum proteins did not rise above 7 per cent. during the fever. In five of these the fever was associated with a

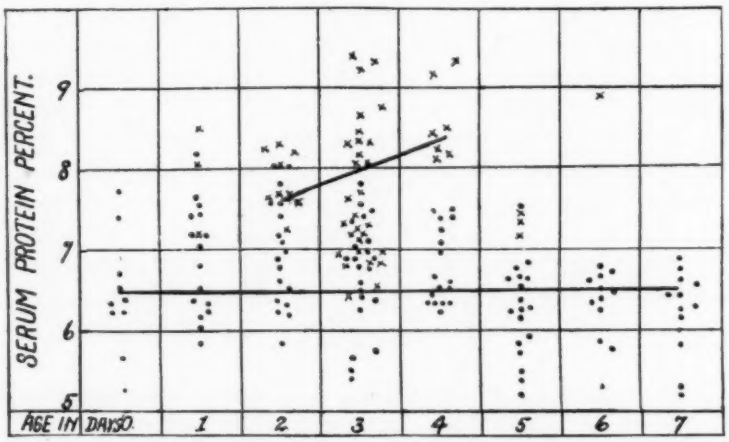

Fig. 1.—The serum protein per cent. in dehydration fever in new-borns. Dots, normal babies; crosses, febrile babies. Upper black line, average for febrile babies; lower black line, average for normal babies.

febrile affection in the mother. It seems possible that a pyrogenetic substance may be transmitted from mother to baby in these cases (Cases 7 and 8). In Case 8 mother and baby had fever at varying times for twenty-seven days after the delivery (Fig. 8). There is fairly close parallelism between rises in the temperature of the mother and baby. The baby's serum protein concentration on the second day of life, when the temperature was 40.7 C. (105.2 F.) was 6.97 per cent., which is within normal limits. In Case 7 the temperature of the mother rose at the same time as the baby's. The baby's temperature remained elevated until it was weaned on the sixth day. In this case also, the serum proteins were not increased.

In Table 1 are shown the weight losses and the serum protein percentages (plasma concentration) of a group of normal and febrile babies. The weight losses in babies with dehydration fever vary from 60 to 700 gm., averaging 350 gm. In the normal babies the weight losses varied from 120 to 540 gm., averaging 280 gm. It will be seen that fever, though more common in babies who had lost a great deal of weight, nevertheless occurred in babies who had lost only small amounts. In these latter babies, however, the increase in serum concentration was as marked as in babies who had lost larger amounts of

TABLE 1.—THE WEIGHT LOSS AND THE SERUM PROTEIN PERCENTAGE IN NORMAL AND FEBRILE BABIES

Normal Babies		Febrile Babies	
Weight Loss, Gm.	Serum Protein, per Cent.	Weight Loss, Gm.	Serum Protein, per Cent.
		700	9.40
		660	8.96
		660	8.37
		620	8.49
		600	7.84
540	7.43	540	8.30
		510	8.61
450	6.22	480	7.73
430	7.31	450	8.89
420	6.39	450	8.02
420	7.33		
390	6.16	360	8.24
390	7.37	360	7.26
330	7.31	360	8.73
310	6.28	330	7.14
300	6.51	310	7.26
		300	8.89
		300	7.26
280	6.80	210	9.19
270	7.84		
240	5.69		
240	6.86		
200	6.34		
180	6.91	180	7.31
180	6.57	180	8.02
180	7.48	150	8.49
180	6.39		
150	7.09		
120	6.39		
		90	8.13
		60	8.49

weight. Losses of weight, therefore, are of significance in relation to fever in the new-born only as they affect the plasma concentration. Since the water content of the baby varies so widely at birth, the weight loss, except perhaps when marked, gives but a poor index of the water content of the plasma.

The hemoglobin varied from day to day without relation to fever, weight or changes in the serum protein per cent. Though there is no relation between variations in the hemoglobin and fever, it is possible that the high red cell content of the blood of new-born babies, by increasing the blood viscosity and thereby slowing the peripheral blood flow, might tend to diminish heat loss and hence make the occurrence of fever more likely.

Comment.—During recent years a considerable amount of evidence has accumulated showing that a close relationship exists between fever and water shifting. In our studies on fever in the new-born, a large proportion of the cases showed a well marked increase in the serum protein per cent., indicating a diminution in plasma water. The concentration of the serum proteins was often as marked as is seen in infants with acute intestinal intoxication. The drop in temperature was accompanied by a well marked drop in the serum concentration. Though the serum of afebrile babies also became concentrated during the loss of weight which occurs in the first three or four days of life, this was rarely as marked as in the febrile babies. Similar increases in the serum concentration are not found in babies with fever due to various infections. It seems clear, therefore, that the cases of fever in new-borns differ from the fevers due to infections in that they are associated with a diminution in the plasma water.

Confirmatory evidence of the close relationship between dehydration and these cases of fever is found in the readiness with which the fever can be reduced by giving fluids. In a number of instances water or glucose solution, warmed to a temperature above that of the baby, was given by gavage. In the large majority of cases so treated the temperature fell several degrees in from thirty to sixty minutes after the gavage.

It seems probable that other factors contribute to the ease with which new-born babies develop fever. It is not unlikely that the high red cell content of the blood, by increasing the viscosity, slows the rate of blood flow and thus diminishes heat elimination.

In the two cases mentioned infections in the nursing mother were associated with fever in the baby. In these babies the serum protein concentration was not increased. It is conceivable that minor disturbances in the mother which pass undetected may also be factors in predisposing, or in some instances causing, fever in the new-born baby.

SUMMARY OF CONCLUSIONS

1. A group of cases has been described in which fever in the new-born baby was associated with a diminution in the plasma water (increase in the serum protein concentration). The fall in temperature was coincident with an increase in the plasma water.

2. A similar increase in the serum concentration is not found in normal new-borns nor in babies in whom fever is due to other causes.

REPORT OF CASES

The following case reports illustrate the close relationship between fever in the new-born baby and the plasma water. Two cases in which fever in the baby was associated with fever in the mother are also shown.

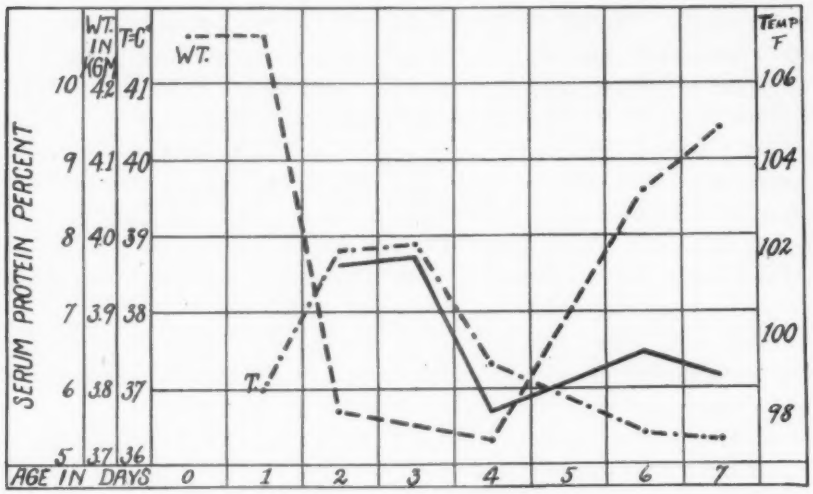

Fig. 2 (Case 1).—The relation between temperature, weight loss and serum protein per cent. (plasma concentration) in dehydration fever. The loss of weight is accompanied by a rise of temperature and an increase in the serum protein per cent. The temperature and serum protein per cent. fall on the fourth day and remain normal thereafter.

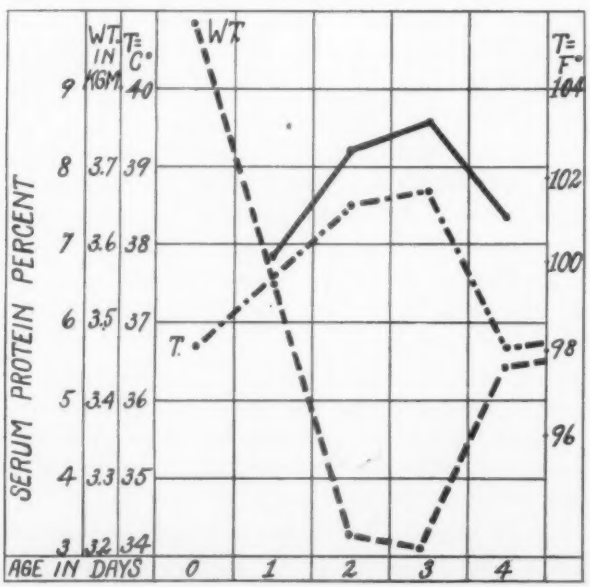

Fig. 3 (Case 2).—The relation between temperature, weight loss and serum protein per cent. (plasma concentration) in dehydration fever. In this case a marked loss in weight is accompanied by a rise of temperature and an increase in the serum protein per cent. The temperature and increased serum concentration persist until the weight gain starts on the fourth day, after which they are normal.

CASE 1 (Fig. 2).—*Dehydration Fever.*—The mother was a primipara, aged 26. Labor was normal. On the afternoon of the delivery the temperature rose to 38.5 C. (101.2 F), but after this it was normal.

At birth the baby weighed 4,260 gm. and was apparently vigorous. During the first two days after birth he lost 490 gm. The temperature, which up to that time had been normal, rose to 38.8 C. (101.8 F.). The patient seemed well otherwise, except that the skin was 2 plus inelastic. The serum protein concentration was 7.61 per cent., indicating a diminution in the plasma water. The fever and hehydration (loss of skin elasticity and increased serum protein concentration) persisted on the third day. On the fourth day, the temperature fell to normal, and at the same time the serum protein per cent. dropped from 7.70 to 5.70 per cent. The baby continued to gain well after this, and the serum protein per cent. remainded normal during the rest of the period of observation.

CASE 2 (Fig. 3).—*Dehydration Fever. Reduction in Temperature and Serum Concentration Following the Administration of Fluids.*—The mother was a primipara, aged 24. The delivery and postpartum course were normal without fever.

The baby weighed 3,880 gm. at birth and seemed to be vigorous. When first observed, twenty-three hours after birth, he had lost 280 gm. The temperature was normal, and there was no evidence of skin or plasma dehydration. The baby continued to lose weight, and by the second day had lost a total of 540 gm. On this day his temperature rose to 38.5 C. (101.4 F.). The skin was slightly inelastic (1 plus), and the serum proteins had risen from 6.86 on the first day to 8.20 on the second. The fever persisted on the third day, the skin and plasma remaining dehydrated. Between the second and third days there was a further loss of 80 gm.

During the night following the third day the patient was given an ounce of 5 per cent. glucose solution, every two hours, in addition to the regular breast feeding. On the fourth day his weight had increased 230 gm. The temperature was normal, and the serum protein per cent. dropped from 8.60 on the third day to 7.31 on the fourth day. The baby continued normally after the fourth day.

CASE 3 (Fig. 4).—*Dehydration Fever Occurring Within Thirteen Hours After Birth.*—This was the mother's second pregnancy. The first child, a girl, was 3 years old and quite well. The postpartum course was normal except for fever and pain in the lower abdomen on the fourth day postpartum.

The patient weighed 3,800 gm. at birth. He was first seen thirteen hours after birth. He had lost 190 gm., the skin was slightly inelastic (1 plus), and the temperature, which was normal at the time of birth, had risen to 38.7 C. (101.8 F.). On the next day the temperature had risen still further, to 40.2 C. (104.2 F.), and the patient had lost an additional 150 gm. By the next day (third day) the baby had gained 90 gm., the temperature had fallen to normal, and he continued afebrile after this. During the fever the serum protein per cent. was increased, falling on the third day with the subsidence of the fever. The baby's temperature rose and fell before the mother's indisposition on the fourth day.

CASE 4.—*Dehydration Fever with Marked Increase in Serum Protein Concentration.*—The mother was a primipara, aged 28. The labor was prolonged and high forceps were applied under ether anesthesia. A large baby weighing 4,430 gm. was delivered. The mother's temperature was about 37.8 C. (100 F.) for the first three days after delivery, after which it rose to 39.5 C. (103 F.). On the fifth day, a small pelvic abscess was opened and drained, and the temperature gradually fell, reaching normal on the twelfth day. The mother left the hospital on the seventeenth day feeling well.

The baby weighed 4,430 gm. at birth. During the first three days after birth he lost 550 gm. His temperature, which up to the third day had been normal, rose to 39.7 C. (103.4 F.). The skin was very loose, but not inelastic.

The serum protein concentration was 9.10 per cent., rising later in the day to 9.40. The baby continued to lose weight between the third and fourth days, losing a total of 700 gm. The temperature was still elevated (39.2 C.) and the serum proteins were 9.30 per cent.

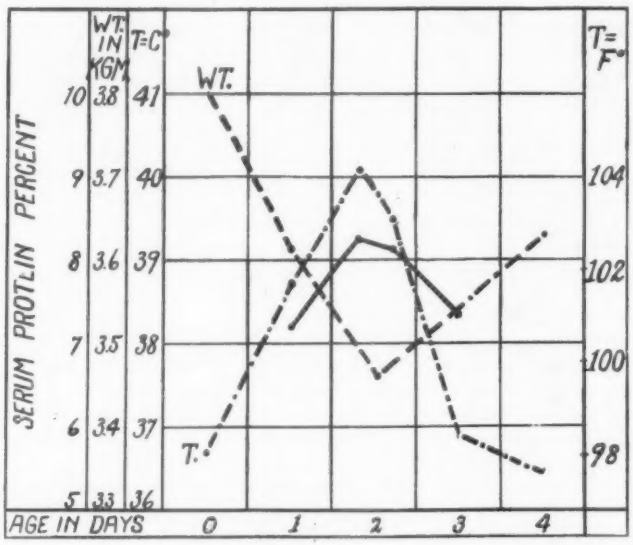

Fig. 4 (Case 3).—The relation between temperature, weight loss and serum protein per cent. (plasma concentration) in dehydration fever. The loss of weight is accompanied by a rise of temperature and an increase in the serum protein per cent. The temperature and serum proteins fall on the third day as the weight gain starts. In this baby the fever occurred within thirteen hours after birth.

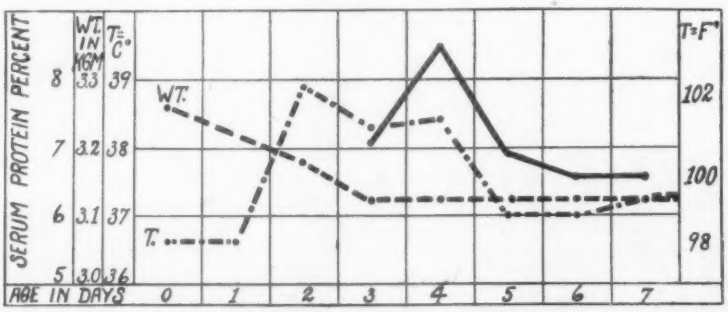

Fig. 5 (Case 5).—Dehydration fever in a new-born baby who has lost only 90 gm. The loss of a few grams is accompanied by a rise of temperature and an increased serum protein concentration. The temperature falls at the same time as the serum proteins.

On the fifth day the temperature dropped to 38 C. (100.4 F.). The serum proteins fell to 8.50 per cent. and the weight increased 60 gm. On the sixth day the temperature fell to normal, and remained there during the remainder of the baby's stay in the hospital. The serum proteins also fell from 8.50 on the fifth day to 7.30 on the sixth day, after which observations were discontinued.

CASE 5 (Fig. 5).—*Dehydration Fever Unassociated With a Large Weight Loss.*—The mother was a primipara, aged 22. Delivery was instrumental (median forceps). The mother's temperature was normal until the fifth day postpartum, when it rose to 38.3 C. (101 F.), after which it remained elevated until the thirteenth day. It then dropped to normal and remained there until the mother's discharge from the hospital on the twentieth day postpartum.

The baby weighed 3,260 gm. at birth. Nothing abnormal was noted until the second day after delivery, when the baby's temperature rose to 38.9 C. (102.2 F.). He had lost only 80 gm. The patient was first seen on the following day (third day). He had lost an additional 60 gm. (total loss, 140 gm.); his temperature was 38.3 C. (101 F.), and the skin and plasma were dehydrated (serum protein per cent., 7.09). On the next day the fever persisted, the serum protein per cent. rising to 8.49. Between the fourth and fifth days, the temperature and serum protein per cent. fell to normal. There was no increase in weight. The subsequent course was normal.

CASE 6 (Fig. 6).—*Severe Dehydration Fever. Fall in Temperature and Serum Concentration Following the Administration of Fluids.*—This was the mother's third pregnancy. Delivery and postpartum course were normal.

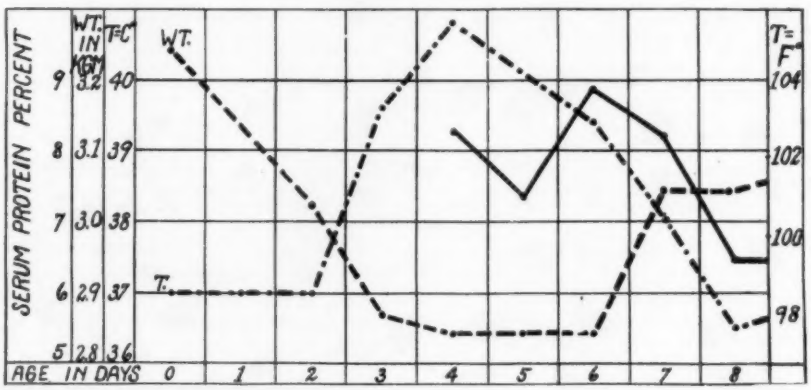

Fig. 6 (Case 6).—The relation between temperature, weight loss and serum protein per cent. (plasma concentration) in a severe case of dehydration fever. This case, like the others, shows the rise of temperature and increase in serum protein per cent. during the weight loss after birth. The serum proteins fall to normal as the temperature drops between the sixth and seventh days.

The baby weighed 3,240 gm. at birth. On the third day, after a loss of 370 gm., he developed fever which persisted until the eighth day, when it returned to normal. On the fourth day, the temperature was 40.8 C. (105.4 F.). During the febrile period the serum protein per cent. was high—8.89 on the sixth day. At this time, water was given by gavage (190 c.c.), and the nurses were instructed to give the baby water frequently during the night. On the following day (seventh day), the temperature fell to 38 C. (100.2 F.), and on the eighth day it was normal. With the fall in temperature the serum protein concentration dropped to normal, remaining there during the rest of the period of observation. The skin, which was inelastic during the fever, returned to normal, when the weight gain started between the sixth and seventh days.

CASE 7 (Fig. 7).—*Fever in the Baby Associated with a Febrile Affection in the Mother. Intermittent Fever in the Baby for Twenty-Seven Days After Birth. No Increase in Serum Protein Concentration.*—The mother was a primipara, 21 years of age. Labor was normal. On the afternoon of the delivery her temperature, which had been normal in the morning, rose to 38.5 C. (101 F.), and remained elevated, with slight remissions, for thirty-four days,

fluctuating between 36.8 C. (98.2 F.) and 39.1 C. (102.4 F.). She was discharged cured on the fortieth day postpartum. The diagnosis was phlebitis of the left femoral vein.

The baby weighed 3,900 gm. at birth. The temperature taken shortly after birth was 37.8 C. (100 F.). It gradually rose and on the second day was

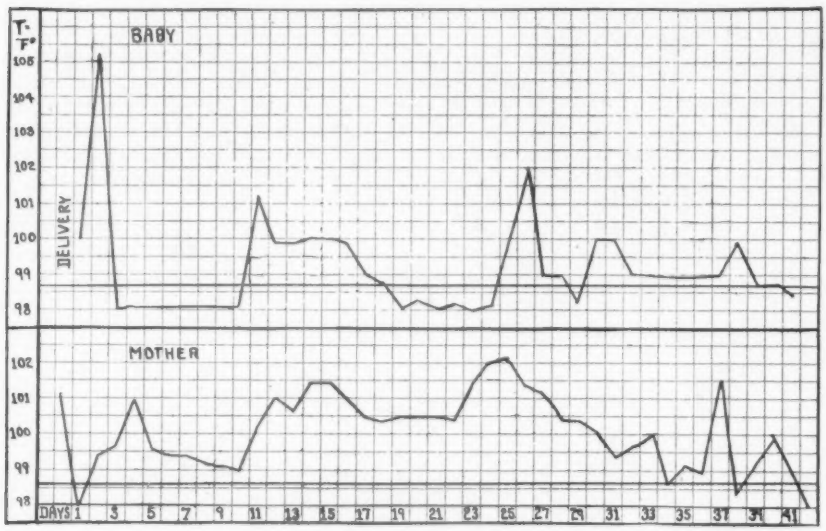

Fig. 7 (Case 7).—The relation between temperature in the mother and temperature in the baby. The upper line represents the temperature curve of the baby, the lower the temperature curve of the mother. Note the parallelism in the two curves. In this baby the serum concentration, determined on the second and third days, was normal.

TABLE 2 (CASE 8).—FEVER IN THE BABY ASSOCIATED WITH A FEBRILE AFFECTION IN THE MOTHER

Baby						Mother	
Age, Days	Weight, Gm.	Temperature, C.	Serum Protein, per Cent.	Skin Elasticity	Remarks	Temperature, C.	Remarks
0	3,730	....	....	.....	.....	36.8	Pelvic abscess
1	.....	37.8	....	Normal	Jaundice	37.1	Endometritis
2	3,410	36.9	5.75	Normal	Jaundice	37.8	
3	3,440	37.2	5.84	Normal	Jaundice	40.5	
4	3,350	38.5	6.97	Normal	Jaundice	41.5	
5	3,300	39.1	6.39	Normal	Weaned	40.5	
6	3,410	36.4	6.86	Normal	Jaundiced	40.0	
7	3,350	36.6	6.34	Normal	Jaundiced	39.0	
8	3,320	36.6	6.28	Normal	Jaundiced	38.5	
9	3,300	37.4	6.62	Normal	Jaundiced	38.5	

40.7 C. (105.2 F.). By this time the baby had lost 180 gm., but the skin was normally elastic and the plasma showed no evidence of dehydration (serum protein per cent., 6.97). On the third day the temperature dropped to 38 C. (100.2 F.), the skin and plasma remaining as observed (serum protein per cent., 6.91). The temperature of the baby and mother continued to fluctuate for thirty-four days, during most of which time the baby gained well. Note the parallelism between the mother's temperature curve and the baby's (Fig. 7).

CASE 8 (Table 2).—*Fever in the Baby Associated with a Febrile Affection in the Mother. No Increase in the Serum Protein Per Cent. Accompanying the Fever. Fall in Temperature on Weaning.*—The mother was a primipara, aged 24. There was no fever until the third day postpartum, when the temperature rose to 40.5 C. (105 F.), remaining at this high level with slight remissions for five days, after which the temperature continued at a lower level. The diagnosis was pelvic abscess, for which the patient was operated on.

The baby weighed 3,730 gm. at birth. His temperature was normal for three days after birth, during which time he lost 390 gm. On the fourth day, one day after the onset of fever in the mother, the baby's temperature rose to 38.5 C. (101.8 F.). It remained elevated until the baby was removed from the breast on the sixth day, after which it was normal for the rest of the baby's stay in the hospital. The serum protein per cent. did not rise above 7 per cent. during the two febrile days. There was no drop in serum proteins when the temperature returned to normal on the sixth day. The skin was normally elastic during the febrile period.

THE TREATMENT OF ACCESSORY NASAL SINUS DISEASE IN CHILDREN *

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OMAHA

The accessory nasal sinuses in children vary in their relative size and development. Schaeffer's [1] measurements in a series of cases show that the antrum is by far the largest cavity at birth and that it retains this relative size in regard to the other nasal sinuses. In my experience it has been the seat of infection much more frequently than have the other nasal sinuses.

Attention has been drawn to infection of the nasal sinuses in children by one or several of the following symptoms: Profuse nasal or postnasal discharge, cough, persistent mouth breathing after tonsil and adenoid removal (Case 1), pale face and lips, running ear, otitis externa, mastoiditis [2] poor appetite, recurring bronchitis,[3] pneumonia and asthma, (Case 5), pleuritis, headaches, loss of appetite, mental hebitude, chorea and nervousness, arthritis [4] chronic pharyngitis, facial acne, conjunctivitis, both phlyctenular and interstitial keratitis [5] swelling of the cervical lymph nodes.

The diagnosis can be made from:

1. *The Discharge.*—In any case of profuse purulent or mucopurulent discharge from the nose sinus infection will account for the discharge. If the discharge cannot be demonstrated—from the history and from the inspection of the nasal cavities and the posterior pharynx—the post-nasal spray will often expell large quantities of secretion from the nose and throat, or these secretions may be brought into evidence on the administration of ether for tonsil and adenoid operations. Atrophy of the nasal mucosa, with glazed sticky scant secretion, usually indicates a long standing antrum infection with involvement of the bone.

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* Received for publication, May 26, 1922.

* From the Department of Otology University of Nebraska Medical Department.

1. Schaeffer, J. P.: The Nose and Olfactory Organs, Philadelphia, P. Blakeston Sons Company, 1920.

2. As a routine I have roentgenographed the sinuses in cases of mastoiditis and have usually found an antrum condition present.

3. Mullin, W. V.: Ann. Otol., Rhinol. & Laryngol. **30**:683 (Sept.) 1921.

4. Dean and Byfield: University of Iowa Studies **2**:15 (April 15) 1921.

5. I have found marked opacity of the antra on roentgen-ray examination of syphilitic children suffering from interstitial keratitis.

2. *Signs of General Toxemia.*—The signs of general toxemia are too well known to be enumerated, but when present, infected nasal sinuses should be considered as among the most likely source of the toxin.

3. *The Roentgenogram.*—The roentgen ray is of great value as a diagnostic aid in antrum infections. In children, however, a dense shadow rarely shows, but a slight density greater than is normal has decided diagnostic value. In few of these cases should an operation be performed, and the roentgen-ray findings are mainly valuable in indicating that treatment should be instituted. As the treatment I have outlined is the same no matter which sinus is affected, no particular localization is necessary. The roentgenograms are also useful to show the progress being made as the result of treatment.

4. *Puncture and Irrigation.*—These procedures are useful in institutions but they are impracticable and quite dispensible in office practice.

5. *External Evidence,* such as broadening of the nasal bridge, and a *skin eruption* over the nasal bridge sometimes may be present.

6. *The pharynx* posterior to the tonsils is often fiery red or covered with mucus or has a thickened mucous membrane with enlarged follicles.

As we recognize the prevalence of antrum disease in children, and as we appreciate its far reaching secondary results, we are impressed with the necessity of working out a satisfactory treatment other than operative or one necessitating prolonged hospitalization. As my patients were mostly office patients, the riot which ensued on any attempts to carry out any effective treatment was so appalling that for some time I contented myself with supplying washes and instillations to be used by the parents in the less exciting environment of the home. However, I found these methods to be exceedingly unsatisfactory and unreliable. I then started to train these children to allow me to use the postnasal spray with compressed air. I found that after winning their confidence and applying the spray to their parents as an example of its painlessness, that after two or three sessions I was able to wash out the nasal fossae thoroughly from the postnasal fossa. This I consider the very best way of cleansing the nasal fossae and also of freeing the ostea of the antra. One has only to see it applied to these little ones to realize its efficiency in expelling large quantities of mucopus from the throat and nose.

There is a great advantage of the postnasal spray over douches and anterior nasal syringing in that I have never once seen any of the wash or infected secretion driven into the middle ear through the eustachian tube. Any spray applied through the anterior nares, if it is to be of sufficient force and volume to clear the nasal fossae and pharynx, will cause considerable trauma and bleeding, thus defeating

its aim. With the postnasal spray the only bleeding is from the pharynx and this only when adenoids or granulation tissue are present. This bleeding rarely takes place after the first two or three applications and seems to be beneficial in relieving the congestion of the nasopharynx.

The postnasal spray should be applied as frequently as is necessary to keep the head clear and to allay the general symptoms. This application should be made daily in some cases and in others only once or twice a week. The average is every other day for about a month and then less frequently. The procedure is of such a simple nature that an inexpensive apparatus is provided for the parents and they are instructed in its use. This, I think, is the proper solution of the problem in many cases. There are so many of these mild antrum conditions prevailing which demand treatment if the children are to reach their proper development, that rhinologists and pediatrists would be swamped with these patients. The parent has only to see the increased well being resulting from a few weeks of this treatment to be willing to provide the means of continuing the treatment himself at home.

Any good alkaline and antiseptic solution can be used. I have for many years used the following modification of Dobell's solution as a nasal wash: Sodium biborate, sodium bicarbonate, 6 gm. each; phenol, 2.5 c.c.; glycerin, 15 gm.; peppermint water, 100 c.c., and distilled water, sufficient to make 480 c.c. This solution is used freely in an atomizer provided with a No. 56 postnasal tip.

I have used this treatment for the past two years for patients aged from 3 to 18 years. All patients have improved very greatly in that the symptoms and signs were greatly lessened or disappeared entirely for a time. During these periods of remission, the strength and resistance of the child was increased, and on reinfection a few treatments would be sufficient to effect recovery. It is my belief that this treatment will often eradicate the infection in children under 8 years. In children over 8, it is mainly supportive of normal development until the child is sufficiently mature to permit of making a patulous opening from the antrum into the inferior meatus by means of an operation. Only a small percentage of children have the poise to go through with a nasal operation under local anesthesia so that ether will generally be necessary when it is considered advisable to operate. Dean's [6] method is given below.

The exact relationship of the antrum to the inferior meatus is ascertained by a careful roentgen ray study. If the antrum has developed sufficiently low to lie well below the inferior turbinate attachment, the opening is made in the inferior meatus; if the antrum has not descended this far, the opening should be made in the middle

6. Dean and Byfield: University of Iowa Studies **2**:7 (April 15) 1921.

meatus. I have found that the opening made in the middle meatus has a tendency to close and that often it is difficult to find it some months after operation. In making the opening from the inferior meatus, the inferior turbinate is turned up out of the way and by means of a bone curet and rasp a sufficiently large opening is made so that later a cannula can be introduced for washing. The inferior turbinate is now turned back to its proper place. If the inferior turbinate covers up the opening so that it would be impossible to introduce a small cannula without pressure, it is advisable to remove a small portion of the anterior end of the bone, as much of the success of after treatment will depend on the possibility of painless washing of the antrum. It must be emphasized to the parent that the operation will not cure the condition but only render the antrum accessible to treatment, and that in order to effect a cure the patient should be under periodic treatment for months and under observation for at least a year.

Instillations of solutions of argyrol, 15 per cent.; phenol in glycerin, 2 per cent.; chloramine T in glycerin or 1 per cent.; dichloramine T in chlorcosane, 1 per cent., are useful whether made in the nose after the postnasal spray treatment or injected directly into the antrum as postoperative treatment after the antrum has been opened.

Dean [7] advises the use of suction by means of Haskins' nasal catheters.

The chronic involvement of the sinuses always originates from the lack of complete resolution of an acute infectious disease. It seems, therefore, that under prophylaxis, we should consider especially routine local treatment of the accessory nasal sinuses following the acute exanthemata. The general relief following the recovery from measles, scarlet fever, etc., is so great that little attention is paid to a remaining purulent condition of the nose, and the acute condition is allowed to become subacute or chronic. A few applications of the postnasal spray during the existence of the exanthemata and during convalesence would do much to prevent the development of so many cases of chronic accessory nasal sinus disease.

Many of these cases clear up simply by removal of the adenoids which probably act mechanically by preventing nasal breathing and causing retention of the infected nasal secretions. Dean [8] has found that diseased tonsils will keep active a sinus infection which may disappear on their removal. Parents must be warned, however, that removal of the adenoids and tonsils will not always clear up an existing infection and that the child should be observed for a year after the operation. Many children who are operated on for return of adenoids

7. Dean and Byfield: University of Iowa Studies **2**:8 (April 15) 1921.
8. Dean and Byfield: University of Iowa Studies **2**:10 (April 15) 1921.

are really suffering from accessory nasal sinus disease and the condition only clears up when treatment is directed to the sinuses.

REPORT OF CASES

CASE 1.—M. K., aged 3½ years, was brought to me with a history of catching cold easily, and of doing a great deal of coughing. Her family physician who referred her to me, had advised the removal of adenoids and tonsils.

Examination.—The child was of normal size and weight but was a mouth breather, and coughed a great deal. The skin below the lid was blue in color. The tonsils were greatly hypertrophied. Much mucous secretion came from the nose but the nares were sufficiently patulous for breathing.

Treatment.—The tonsils and adenoids were removed under ether anesthesia. During the operation it was noted that there was profuse secretion from the nose into the throat.

Course.—The cough was relieved to a great extent for three weeks when it returned, keeping the child and parents awake the greater part of the night. A second examination showed that the pharynx was fiery red and that it was thrown into vertical folds on gagging. The nose was partly filled with mucopus. The child was pale, tired, nervous and fretful.

A diagnosis of sinus disease was made. This was verified by roentgen-ray examination. The left antrum was cloudy and the right antrum was slightly cloudy.

Postnasal spraying was commenced and a large quantity of mucus was expelled through the nose. The results were immediate. The child slept through the night for the first time for a week. After the second treatment, the child was entirely tractable and did not cry or resist the spraying. Gradually her appetite became good, her color improved and she went through the rest of the winter without further bronchial trouble.

CASE 2.—F. M., aged 13, when seen by the family physician two weeks ago, had a temperature of 102 F., suffering with severe pain in the left cheek. There was a great deal of discharge from the nose, particularly from the left nostril. He had never had an attack like this before, but was always subject to colds. His physician made a diagnosis of antrum infection. This diagnosis was verified by the roentgen ray which showed the right antrum almost closed, the left antrum completely blocked and the other sinuses in a questionable state. The middle turbinates were boggy and swollen. His parents stated that for several months he had not been as robust as formerly, and that he had been subject to bronchial attacks.

Treatment.—After three months of postnasal spraying he regained his normal strength. Another roentgenogram taken at this time showed the right antrum one plus, and the left antrum one and one-half plus. The other sinuses were clear. He had not had any bronchial attacks during the variable weather prevailing through the season he was under treatment. He is apparently in robust health. He should be kept under observation and treatment, when necessary, for the next two or three years as the roentgen ray shows still some latent infection.

CASE 3.—S. R., boy, aged 9. His mother states that he has not been well for the past four years. He is listless, has a poor appetite, is pale and somewhat small for his age. His nose discharges summer and winter. Adenoids were removed three years ago.

Examination.—The tonsils were small; pus was expressed fairly easily. Mucopus was dropping from the posterior nares into the nasopharynx in large globules. The teeth were clear. Mucopus was present in both middle fossae of the nose.

Diagnosis.—Nasal sinus disease; mild infection, verified by roentgen ray which showed the right antrum to be cloudy, the left antrum slightly cloudy, and the other sinuses clear.

Treatment.—This consisted in postnasal spraying three times a week at the office and a nasal wash at home. Errors in diet and hygiene were eliminated.

Course.—The boy showed marked improvement from the start and in two months he was a normal healthy boy with red cheeks and a good appetite, and he was doing well in school. At this time it was decided to remove his infected tonsils. The sinus infection is not cured but it is controlled so that he is going on to healthy development.

CASE 4.—F. D., boy, aged 7, when first seen was in bed, with a high temperature, fiery red pharynx and mucopus in the nose. The tonsils and adenoids had been removed. The boy had a history of constant poor health, nervousness and repeated colds.

Treatment was given for the acute condition and on recovery roentgen-ray examination showed all sinuses slightly cloudy. The boy was taken out of school, given outdoor life and supportive treatment, and treated regularly with the postnasal spray at the office.

His general health became normal and he went through the winter without colds and with a steady gain. His family left this city and instruction was given to continue the treatment as the local condition was under control but not cured.

CASE 5.—R. E., boy, aged 7, has had asthma for two years. The attacks last longer than formerly but they are less severe. His tonsils and adenoids were removed one year ago, but he did not get much relief.

Mucopus was present in nose and nasopharynx. The bases of the tonsils remain. A roentgenogram taken four months previously showed both antra cloudy; the other sinuses were clear. The child is pale and nervous.

Treatment.—As the parents lived at some distance and the asthma was becoming established, and was of long standing, it was considered better to make an opening beneath the inferior turbinates according to Dean's method. The remains of the tonsils were removed, and some adenoid bands were broken up. The parents were instructed in the use of the postnasal spray and before the child returned to his home he was trained to submit to treatment.

Course.—He went through the winter without any asthma, gained in weight and has good color. There is still mucopus in his nose and throat occasionally and at times he needs the postnasal spray. It is too early yet to say that the asthma may not return but the effect of the operation and treatment have been so far successful.

The study of sinus disease in children only dates back a few years and our ideas on all phases of this important subject have yet to be crystalized. It is with the purpose of arousing interest and further observations by others that I have submitted this brief paper.

A NEW ERUPTIVE FEVER ASSOCIATED WITH STOMATITIS AND OPHTHALMIA

REPORT OF TWO CASES IN CHILDREN *

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During a period of three months we had the opportunity of observing two cases of an extraordinary, generalized, eruption with continued fever, inflamed buccal mucosa and severe purulent conjunctivitis. The first patient was seen on the tenth day of illness and followed to recovery. The second patient did not come under observation until the twenty-second day after onset of the illness; but the skin lesions at that time corresponded exactly with those of the first case at the same stage of the disease, and a careful description of the eruption as seen in the first week establishes its identity with that in Case 1.

The condition was so unusual and so entirely unlike anything previously observed, that pains were taken to get as many expert opinions as possible from men of wide clinical experience. At the same time, a search was made in the literature of eruptive fevers and allied dermatologic conditions. No diagnosis could be made to correspond with the symptoms and course of the eruption in these two cases and no description was found of a skin condition in any degree comparable.

Figures 1, 3, 4 and 5 will make clear, as verbal description cannot, the nature of the eruption in its several phases. Figure 1 shows the boy on the thirteenth day of his illness. The eyes are swollen shut, exuding pus; the lips are black with crusted blood. The eruption is complete, except for the feet and hands, while on the shoulders, scaling of the horny crusts is already in progress. Figure 3 shows the same boy on the twenty-fourth day. Many of the lesions on the back and chest have already crusted and dropped off; the later lesions of the feet and hands show clearly, but on the arms and thighs there is an exaggerated effect of confluent desquamation due to the liberal use of bland ointments and powder on the skin. In this detail the photograph is somewhat deceptive. Figure 4 shows the same boy on the thirty-third day of his illness. The mouth and eyes are normal; the face, chest and back are practically clear, except for pigmented spots which mark the site of lesions which have crusted and fallen away. Figure 5 shows the other boy (Case 2) eleven weeks after the onset of his disease.

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* From the Department of Diseases of Children, Columbia University and the Children's Medical Division, Bellevue Hospital.

The sunken, sightless eyes bear witness to the destructive effect of the ophthalmia in this case. Over the whole body may be seen the brownish pink pigmented spots that mark the position of the eruption.

In the uncertainty of diagnosis, each case was treated in isolation with the strong impression in the minds of all that this disease, with its high temperature and profuse eruption, should be considered in the group of infectious, eruptive fevers. There was an interval of a litttle more than a month between the arrival of the patients at Bellevue Hospital and no possible contact could be traced between the families—living, as they were, in widely separated sections of the city. At any rate, no other cases developed; and while the presumption remains that this condition is an infection, there is nothing to indicate that it is spread by immediate contagion.

Fig. 1.—Case 1. Nature and extent of eruption on the thirteenth day of the illness.

REPORT OF CASES

CASE 1.—*History.*—H. S., a white boy, aged 8 years, was admitted to the children's service of Bellevue Hospital, May 1, 1922, on the tenth day of his illness. There was nothing significant in the family history; he had diphtheria at 18 months, pertussis at 3 years and measles at 7 years. He had been successfully vaccinated and the tonsils had been removed at 7½ years. His general health had been good until shortly following the tonsillectomy when he was in a hospital for fourteen weeks with a septic temperature, enlarged spleen, jaundice and a blood culture which showed *Staphylococcus aureus* on two occasions. He recovered from this, and was apparently well for two and a half months prior to the onset of the present illness.

Onset of Illness.—Ten days before admission, he complained of a mild sore throat and general malaise. He was not very sick at first, but felt weak and went to bed. The next day he complained of pain in the eyes and did not want to eat because his mouth hurt him. Soon pus was seen running from the inflamed eyes and on the third day reddish spots were noticed on the back of the neck. The body was not examined until the seventh day when the

skin was found to be covered with reddish brown spots; only a few such spots were noted on the face, legs and arms. While at home, the temperature was not taken.

Physical Examination.—On admission the boy was acutely ill with a temperature of 103 F. He was well nourished, mentally clear and cooperative, but was prostrated and unable to open his swollen eyes. The lids of both eyes were edematous and thick pus streamed down the face from the palpebral fissures. There was marked blepharitis marginalis, chemosis and inflammation of the bulbar conjunctiva with haziness of the left cornea. The lips were cracked, bleeding and encrusted. The tongue was swollen, bright red and

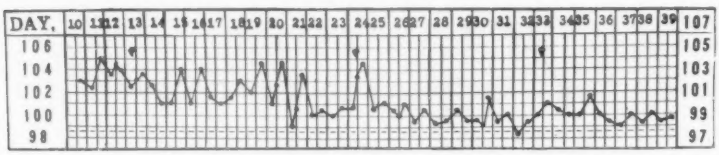

Fig. 2.—Temperature chart of Case 1. P marks the days when photographs were taken.

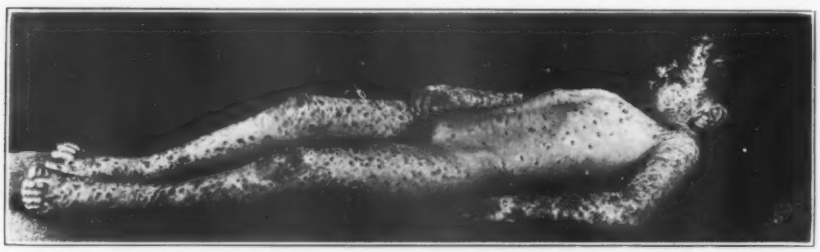

Fig. 3.—Case 1. Twenty-fourth day of the illness.

Fig. 4.—Case 1. Thirty-third day of the illness.

fissured, and the mucous membrane of the mouth was inflamed with small bullous lesions which rapidly broke down, leaving a raw and angry surface. The scalp was quite free of any eruption but the face showed large blotches of dried pus, considered secondary to the purulent discharge from the eyes spreading over the papular lesions of the cheeks and forehead.

The trunk, arms and thighs were thickly set with discrete, oval, brownish purple papular lesions, varying in size from 0.5 to 2 cm. in the longest diameter. A few of the largest spots showed a yellowish, dry, necrotic center. There was no vesicle, nor pustule formation; no induration about the papule; no areola and the skin between the lesions seemed to be perfectly normal. The

forearms and legs showed what seemed to be the more recent lesions. These were less raised, of brownish red color, paling somewhat on pressure, and more closely set than were those of the trunk and thighs (Fig. 1).

Careful physical examination disclosed a faint short systolic heart murmur, heard best to the left of the apex and not transmitted. The spleen was palpable 4 cm. below the ribs, and the liver edge could be felt a finger's breadth below the costal margin. Otherwise, there were only normal findings.

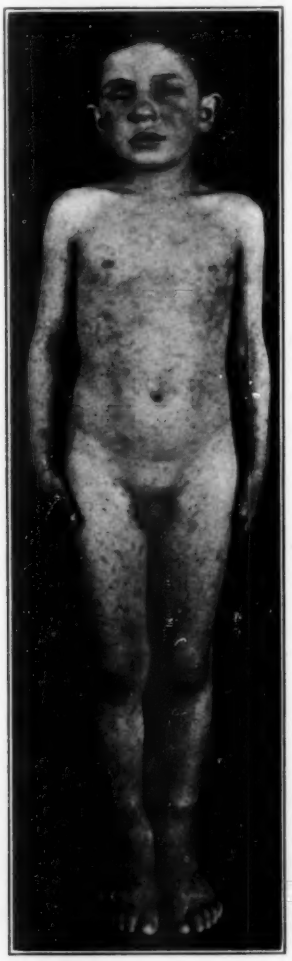

Fig. 5.—Case 2. Eleven weeks after onset of illness. Recovery was complete except for total blindness. Persistent pigmentation shows at the sites of the skin eruption.

Laboratory Examination.—Cultures made from the eyes and throat showed a mixed infection of common cocci and bacilli with no predominating organism and no gonococci. From the necrotic center of one large papule a pure strain of *Staphylococcus aureus* was grown. The blood culture was sterile. The Wassermann reaction was negative.

Blood: The blood count showed 4,000,000 red blood cells with 75 per cent. hemoglobin, and 10,000 leukocytes with 82 per cent. polymorphonuclears and 18 per cent. lymphocytes and transitionals. The blood chemistry was also done and all values were determined to be within the normal range.

Urine: Except for a trace of albumin, which was a constant finding during the period of high temperature, the urine was negative.

Course.—For the next eleven days (from the tenth to the twenty-first day of the illness) the temperature ranged between 101 and 105 F., with a fall to the 100 degree level after the twenty-fourth day (Fig. 2). At each examination the blood showed a definite leukopenia.

During this time there was no complaint of pain or itching or discomfort in the skin lesions. Photophobia and pain in the eyes persisted, but the edema of the eyelids and the purulent discharge yielded to treatment with warm boric acid instillations, dilatation of the pupils with 1 per cent. atropin and the application of 1:5,000 mercuric chlorid in petrolatum twice daily. The right eye cleared rapidly, but the left cornea was already ulcerated and it healed very slowly, with resultant scarring of its lower segment.

New lesions were seen to appear on the skin of the forearms and legs, the palms and soles, for the first week after admission, but none after the eighteenth day since onset. These were at first pale and macular, rapidly becoming firm, dry, purplish papules like those seen on the trunk at the time of admission. Meanwhile, the earliest skin lesions had begun to resolve into

Blood Examination in Case 1

	19th Day	26th Day	37th Day
Leukocytes	4,600	6,700	8,700
Polymorphonuclears	62	52	58
Small lymphocytes	33	45	40
Large mononuclears	3	0	0
Eosinophils	0	2	2

horny, flattened, thick scales, with raised, papery edges. At this stage the fever dropped to its lowest level (Fig. 3).

As the resolution advanced in the same order as the appearance of the lesions, these dry horny scales dropped off from the neck and back, then from the chest and abdomen and thighs, and last of all from the arms and legs, leaving a dull reddish area of apparently normal skin to mark the site of the lesions. There was no instance of pitting or scarring of the skin (Fig. 4).

The inflammation of the mucous membrane of the mouth, lips and tongue subsided within a week of admission. This did not interfere with the taking of nourishment and no gastro-intestinal symptoms were present at any time.

The subsequent history of this case is not especially significant. After a period of normal temperature for two weeks a left otitis media developed; this went on slowly to a mastoiditis for which the boy was operated on June 22. At this time a few crusts still remained on the forearms and feet.

Case 2.—*History.*—S. M., was a Jewish boy, aged 7 years, with a negative family history, who had been always strong and well except for measles at 4 years. He had been vaccinated successfully.

Onset of Illness.—Three weeks before admission, he complained of pain in the eyes and weakness. The next day the eyes were discharging, and a rash was noted on the body which the family physician called "black measles." On the third day of his illness he was taken to the Willard Parker Hospital, where he was found to have a temperature of 103 F., with signs of bronchopneumonia at the right base, a purulent conjunctivitis with great edema of the lids, and a skin eruption of which the tentative diagnosis of erythema multiforme was made. This eruption was described on the third day as being macular, hemorrhagic, with a few papules, with areas of excoriation on the face.

The eyelids were edematous and pasted shut with thick pus, and the mucous membrane of the mouth was practically exfoliated. The boy remained at Willard Parker Hospital for two and a half weeks, with the pneumonia resolving gradually. The temperature ranged from 101 to 104 F. for two weeks, then became irregular at a lower average with an occasional rise to 103 F. The boy was irrational during the first week, and there was great prostration, weakness and loss of flesh. In spite of instillations of boric acid solution and argyrol, corneal ulcers formed, with perforation and abscess formation in both eyes involving all the structures of the globe.

Simple ointments were applied to the skin lesions, which crusted and dropped off the chest and back by the end of the third week.

He was transferred to the Children's Service of Bellevue Hospital on the twenty-second day of his illness, March 11, 1922.

Physical Examination.—On admission the boy was acutely ill, much wasted, with a temperature of 102 F., mentally clear, but totally blind. The lungs showed signs of a resolving pneumonia of the right lower lobe. The eyes were the seat of a panophthalmitis, with destruction of the cornea and lens. Pus was still oozing from the granulating tissue of the shrunken eyeballs.

The skin of the trunk was the seat of irregularly oval pigmentation of a reddish brown color, rather evenly distributed except below the nipples where there were confluent areas. This skin was quite normal in appearance, except for the pigment, and showed no trace of scar tissue.

The arms and legs showed oval brownish red horny crusts, varying from 1 to 2 cm. in the longest diameter without areola or surrounding induration, and without trace of exudate or pustule formation. There was no complaint of pain or itching. Otherwise the examination was quite negative.

Course.—The lung condition cleared slowly with a temperature varying between 101 and 103 F., for a week, then falling to the 100 degree level for eight days. The eyeballs healed by granulation. The skin condition resolved progressively by the falling off of the thick oval horny crusts from the thighs and arms, and, last of all from the feet and soles, each crust leaving to mark its site an area of reddish brown pigment. These areas were identical with the marks found on the back and chest at time of admission.

The family refused to allow blood to be taken for a Wassermann test and insisted on removing him from the hospital fifteen days after admission. Later, this boy was visited in his home and found to be in excellent condition except, of course, for his blindness. With some difficulty consent was obtained for a photograph (Fig. 5).

SUMMARY

1. Two cases have been observed of a generalized cutaneous eruption, not conforming to any recognized dermatologic condition.

2. Both cases occurred in boys, one aged 7, the other 8, coming from widely separated parts of New York City, with no possibility of contact.

3. Both cases manifested a purulent conjunctivitis, in Case 2 going on to panophthalmitis and total loss of vision, and in Case 1 responding to treatment, but leaving a severe corneal scar. The pus showed pyogenic organisms only; no gonococci.

4. A high and continuous fever was present in both cases, explainable in Case 2 by a lobar pneumonia, but in Case 1 without apparent cause other than the skin condition.

5. The eruption showed certain characteristics, identical in each case. The onset was with fever, the rash appearing on the back of the neck

and chest, spreading to the face, arms and legs during a period of about eighteen days, the last lesions to appear being on the soles and palms. At this time resolution of the first lesions began.

The eruption consisted of oval, dark red to purplish macules, separated by normal areas of skin. These became in a few days raised firm papules of brownish purple, from 0.5 to 2 cm. in the longest diameter, without areola, and without subjective symptoms of pain or itching at any time. A few of the largest spots showed a yellow, dry, necrotic center. The lesions on the forearms and shins were smaller and more thickly crowded together. No pustules or vesicles were to be seen. The scalp was at all times free of lesions; but the mouth and lips were intensely sore and inflamed. In Case 1 bullae were noted in the mouth at the end of the first week.

After the third week resolution began in the order of appearance of the lesions. This consisted of a shrinking of the macule to a horny oval of dark brown color, with raised papery edges. From the fourth week these scales dropped off, leaving a faint pigmented area, without pitting or scarring. By the fifth week the chest, face and back were clear, except for pigmentation, while resolution and crusting were still going on on the forearms and legs. Fall of temperature coincided with the period of resolution of the skin lesions.

DIAGNOSIS

1. A skin eruption from drug ingestion is ruled out by careful inquiry which showed that no drugs whatever had been administered in either case.

2. A skin eruption from food poisoning can hardly be considered from the entire absence of gastro-intestinal symptoms in the presence of a high and sustained temperature reaction.

3. Syphilis is to be excluded by the character of the lesions, the age of the children, the negative history, and the negative Wassermann reaction in Case 1.

4. Pemphigus was suggested, from the observation of bullae in the mouth in Case 1. Nothing further in the course of the disease, the appearance of the eruption and its evolution gave any support to this diagnosis.

5. Hemorrhagic measles, as it happened, was the primary diagnosis of the family physician in each instance. Both children had already had measles, in Case 1 at 7 years, and in Case 2 at 4 years. While excusable at the onset, this diagnosis is quite indefensible in view of the distinctive character of the lesion as described.

6. A sepsis with generalized eruption must be considered as a possibility, in spite of the negative blood culture in Case 1. This diagnosis

is suggested by the history of a prior sepsis in Case 1 and might account for the temperature and course and the occurrence of a pneumonia in Case 2. The superficial nature of the lesions, their character and progressive appearance over nearly three weeks and the lukopenia argue against this supposition.

7. Erythema multiforme, from some unknown toxic cause, was proposed as a diagnosis, but this is unsatisfactory from the character and distribution of the lesions, the lack of subjective symptoms, the prolonged high fever, and the terminal heavy crusting.

8. There remains the possibility that this condition represents a distinct disease which has not hitherto been recognized.

Comment.—While the true diagnosis of this condition must be left an open question, the report of these two cases seems justified if only to draw attention to the serious effects to be expected unless the eyes in any similar case receive early and painstaking treatment. The boy in Case 2 is totally blind, while the other boy only escapes with an impairment of vision in one eye. Furthermore, it is hoped that this report may lead to the study of other or similar cases. Quite possibly the observation of this condition in its first stages may clear up the vexed question of its etiology.

Finally, we have been impressed by the striking picture presented by these cases. Here is a syndrome of dramatic onset, with fever, conjunctivitis and cutaneous eruption. The child is prostrated, the mouth and tongue are inflamed and raw, the eyelids are swollen and pus streams from the eyes. There is a course of three or more weeks of high fever, with leukopenia. The eruption, unlike any hitherto described, comes out progressively, for two weeks or more, matures and resolves in horny crusts, in the order of its appearance. The temperature falls with this resolution of the skin lesions. This syndrome suggests strongly an infectious disease of unknown etiology. We believe that this condition deserves to be considered a definite clinical entity.

We wish to express our appreciation of the kindness of Dr. L. Emmett Holt, Dr. John A. Fordyce and Dr. Warfield T. Longcope, who saw one or both of these cases in consultation.

CONGENITAL DUODENAL OBSTRUCTION: MALROTATION OF THE INTESTINE

REPORT OF CASE *

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NEW YORK

In this report the clinical rather than the developmental aspects of congenital duodenal obstruction will be emphasized. It seems that enough cases have been recorded to warrant giving a more definite clinical picture, and this may serve to center interest in them as clinical cases rather than as pathologic specimens. Unfortunately, the first few days of life are a veritable shadowland for diagnosis and treatment. The infant that seems barely holding on to life may be a thriving youngster a few days later, and, on the other hand, sudden and unexplained exitus of a baby apparently not very ill is an all too frequent occurrence. Similarly, at this time of life exploratory laparotomy is an adventure. It is, therefore, with some hesitancy that the effort is made to group the striking clinical features of congenital duodenal obstruction and to suggest that at least some of the cases may be brought within the reach of surgical assistance. Perusal of the literature, in addition to the study of a single case, forms the background for the discussion and conclusions.

From the clinical standpoint, the possible duration of life of infants suffering from duodenal obstruction is of first importance. As far as this is related to the anatomic anomaly, the duration of life is dependent on whether there is atresia (complete occlusion of the intestinal tract) or merely stenosis (partial obstruction). Of thirteen cases of stenosis collected by Kreuter in 1905, four patients lived for a week or more.[1] A search of the literature since 1905, probably not complete, of cases of partial obstruction in which the patients live more than a week, has revealed an even greater number.[2] Four infants died during the second week of life, and three during the second month; four others lived from four to nine months, one for twelve months,

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* From the Pediatric Service of the New York Nursery and Child's Hospital and the Pediatric Department of Cornell University Medical School.

1. Two infants lived seven days, one lived twelve days and one six months.

2. This refers only to infancy and not to those cases of obstruction discovered in later life, due to bands, anomalous peritoneal fossae, malrotations, etc.

one for eighteen months and one for two years.[3] The mere duration of life is emphasized because it indicates that in some cases there is at least time for surgical intervention.

Efforts at treatment have been made. Colwell, in 1912 recorded five cases in which enterostomy had been done, although unsuccessfully. Commenting on the reports of Keith and Buchanan of infants living nine and eighteen months, he remarked, "it would seem that cases should be subjected to laparotomy, and, if possible, gastro-enterostomy should be performed." Indeed, since then such an operation has been done successfully. Ernst, in 1916,[4] operated on an infant 11 days old. There was an obstruction "at the upper side of the transverse mesocolon" and Ernst performed a duodeno-entero anterior anastomosis. Eleven months later the child was a "vigorous well developed youngster."

The symptomatology of cases living more than a week depends on the location and degree of obstruction. When the obstruction is situated below the ampulla of Vater, the vomitus is bile stained. This is emphasized by nearly all the authors who considered the symptomatology of the cases they reported.[5] Bradford believes that "vomiting occurs after the lapse of a longer period following feedings than does the vomiting caused by stenosis higher in the tract." In nearly all cases there was difficulty in excluding pyloric stenosis, and in many cases that was the diagnosis made. Tumor was mentioned by only a few authors; it was a vague sign.[6] Peristaltic waves were more frequently found. Fecal stools, the occurrence of the residue of milk digestion or the continuance of meconium stools beyond the usual period were helpful in determining partial or complete obstruction. Prolongation of life, despite an almost complete obstruction was a surprising

3. Commandeur: Lyon méd. **114**:423, 1908. Freeman, R. G.: Arch. Pediat. **27**:37, 1910. Shaw. H. L., and Baldauf, L. K.: Tr. Am. Pediat. Assn., 1907, p. 36. Weber: München. med. Wchnschr. **60**:212, 1913. McClanahan, H. M.: Arch. Pediat. **25**:533, 1918. Stolte, K.: Monatschr. f. Kinderh. **12**:341, 1913. Colwell, E. M.: Quart. J. Med. **5**:401, 1911. Spriggs, N. I.: Guy's Hosp. Rep. **66**:143, 1912. Van Der Bogert: Arch. Pediat. **33**:194, 1916. Schroder, C. H.: J. A. M. A. **78**:1039 (April 8) 1922. Cautley, E.: Brit. J. Child. Dis. **16**:184, 1919. Bradford, O. F.: J. Missouri M. A. **15**:307, 1918. The case of Helmholz and Little (Johns Hopkins Hosp. Bull. **16**:231, 1905) should be included, although it was a complete obstruction. A gastro-enterostomy was done and the child lived eight days.

4. Ernest, N. P.: Brit. M. J. **1**:644, 1916.

5. A possible exception to this statement is Van Der Bogert's case. In an infant, 4½ months old, with a duodenal obstruction 2½ inches from the pylorus "there was no appearance of bile in the vomitus, although unfortunately no chemical tests were made."

6. Loc. cit. Cautley and also Schroder.

feature. In Cautley's case, in which the child lived twelve months "the second part of the duodenum was extremely stenosed, merely admitting the passage of a probe." And Spriggs, performing a necropsy on an infant 9 months old, found a diaphragm occluding the lumen of the duodenum; and, as he remarked, although there was "no evidence of an orifice in this diaphragm, the history of the case proves that some opening must have existed." The uncertainties of abdominal diagnosis, in general, at this early age, combined with variations depending on the position and degree of obstruction, make it very difficult to outline a clear cut clinical picture. One might be tempted to offer a crude rule of thumb, that a symptom complex suggesting but not typical of pyloric stenosis or pylorospasm, in a desperately sick infant, should raise the question of an obstruction in the upper part of the small intestine.

The etiology of duodenal obstruction has been fully reviewed.[7] Many of the anomalies and the theories to explain them are referable to conditions within the lumen of the intestine, valves, folds of mucous membrane, failure of absorption of embryonic epithelial plugs, etc. The case to be reported was not studied from that angle, and although it is extremely unlikely, such a factor might have been operative in addition to malrotation.

REPORT OF CASE

History.—Baby R. was born on the obstetric service of the New York Nursery and Child's Hospital, Dec. 24, 1920. Toxemia of pregnancy necessitated delivery by cesarean section during the eighth month of parturition. The premature baby weighed 6½ pounds and appeared well developed. When 7 days old the child was transferred to the pediatric service with the history of 1½ pounds loss of weight and vomiting of all food since birth; the vomitus was dark green in color and not projectile.

Course.—Dr. Anderson, the resident physician, noted that the baby was emaciated, the fontanel slightly depressed, the abdomen sunken and the liver and spleen palpable. The infant lived twenty-six days after admission to the pediatric service. Although occasional days passed without vomiting, persistent bilious vomiting was the striking symptom. Lavage on numerous occasions also revealed bile stained fluid, at times as much as from 3 to 4 ounces of "thick pea soup material." At first, the stools were yellow and indicated the passage of food through the intestines; toward the end they became watery and green. The roentgen-ray examination was not conclusive. It showed no peristaltic waves, an atonic stomach and a threadlike shadow outlining the course of the duodenum. Three days before death, definite peristaltic waves were seen. No tumor could be felt. Atropin was administered for a few days. It is hardly necessary to detail the supportive measures employed—saline solution subcutaneously and into the peritoneal cavity, and transfusion—and the care in matters of feeding.

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7. Kreuter, E.: Die Angeborenen Verengerungen u. Verschliessungen des Darmkanals. 1905, St. George, A. V.: Am. J. Dis. Child. **15**:354 (May) 1918, and Cordes, L.: Arch. Pediat. **13**:401, 1901, in addition to those of Spriggs, Cautley, and Helmholz and Little. These refer to atresia as well as to stenosis.

Fig. 1.—The liver has been pulled upward and the transverse colon downward. This gives the impression that pressure occurred lower down in the duodenojejunum than was actually the case. (1) Sharp duodenojejunal angle; (2) jejunum; (3) lower part of ileum; (4) band attaching cecum to posterior abdominal wall.

Diagnosis.—The diagnosis lay between pyloric stenosis and obstruction below the ampulla of Vater. Bilious vomiting, pointing to relaxation of the pyloric sphincter, was the striking symptom and most of those who examined the infant favored the diagnosis of obstruction. A second transfusion preliminary to exploratory laparotomy was planned when the infant died.

Report of Necropsy.—Aside from the intestinal tract, the necropsy is not of interest. The small intestine occupied the entire right half of the abdomen. The cecum was situated to the left of the midline beneath the transverse colon (Fig. 1). Drawing the cecum downward and to the right, brought out a firm, rather tough band, attaching the cecum to the posterior abdominal wall. The ileum entered the cecum from right to left. The stomach was much dilated and the pylorus was wide and flabby. The dilatation of the first portion of the duodenum was even more pronounced than is indicated in the sketch.[8] The third portion of the duodenum, instead of curving gradually upward, turned to the left, creating a sharp duodenojejunal angle. To express these relations

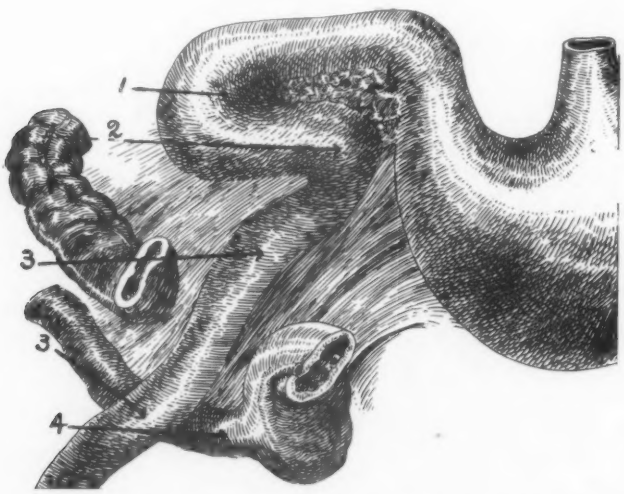

Fig. 2.—Ascending colon cut and pulled back to show mechanism of obstruction. (1) Dilated duodenum; (2) duodenojejunal angle; (3) jejunum; (4) ileum.

differently, the third part of the duodenum was missing and the duodenojejunal angle was formed by the transverse or second part of the duodenum and the continuous loop of the intestine. As this loop of intestine continued downward, it emerged, clamped between the last few inches of the ileum dorsally and the cecum and ascending colon ventrally. One may picture the ileum and the ascending colon as the blades of a scissors (Fig. 2). The posterior or dorsal blade is formed by the ileum, the anterior or ventral blade by the ascending colon, the ileo-caecal region forms their point of attachment and the duodenum is clamped between them. Figure 2 shows the ascending colon cut and rolled back, uncovering the duodenum as it crosses the ileum. Thus, three factors contributed to the production of the obstruction: (1) the sharp duodenojejunal angle; (2) the squeezing of the jejunum between ileum and colon;

8. The sketch was made from the preserved and somewhat shrunken specimen.

(3) the binding down of the cecum to the posterior abdominal wall. The fixation of the cecum prevented free mobility, and a freely movable cecum might have exerted less pressure on the small intestine.

It is simpler to describe the anatomic conditions determining the obstruction than to trace their pathogenesis. The explanation of the mechanism of obstruction rests on the determination of the primary factor in the malrotation of the cecum and ascending colon. It will be recalled that in early fetal life the alimentary canal is a vertical tube. As development proceeds, the colon, which is at first on the left side, rotates about the mesenteric axis and the cecum passes across the duodenum and finally descends to the right iliac fossa. "This rotation may be arrested temporarily or permanently in any position during its circuit."[9] In the case presented there are two possibilities. First, there might have been a localized peritonitis that produced a fibrous band maintaining the cecum in the position characteristic of an earlier period of fetal life, that is, to the left of the median line (Fig. 1). According to this explanation, the fibrous band, or rather the inflammation responsible for it, would be considered the primary factor preventing normal rotation of the large intestine. Or, secondly, there might have been some other unknown primary factor interfering with normal rotation; according to this assumption, the fibrous band would be considered the result of a secondary contraction of a loose peritoneal fold. The sharp duodenojejunal angle was surely secondary; most probably it was due to the drag exerted by the small intestine. The loops of intestine hung from the mesentery as from a pedicle instead of spreading out fan shaped in the normal fashion. In either case failure of the intestine to rotate completely was the immediate cause of obstruction since it left the cecum in such a position that pressure upon the duodenum was unavoidable.

DISCUSSION AND CONCLUSIONS

Duodenal obstruction in infants, and especially in new-born babies, embraces too many variant factors to warrant generalizations. The most that can be expected, as far as diagnosis is concerned, is the determination of the presence of obstruction, whether it is partial or complete, and its approximate location. Recording the symptomatology may develop a clearer clinical picture. The results of surgical intervention depend chiefly on the complexity of the lesions. Surgeons who have seen the specimen doubt very much whether anything could have been done in the case described. In general, gastro-enterostomy is

9. Mayo, C. H.: Med. Rec. **81**:401, 1912.

the operation indicated. The duration of life in some infants with duodenal obstruction and the successful performance of gastroenterostomy by Ernst suggest that under favorable circumstances some cases may be amenable to surgical treatment. The cause of the obstruction in the case reported was malrotation of the intestine; whether the malrotation was primary or due to a fibrous band binding the cecum to the left abdominal wall, is controversial.

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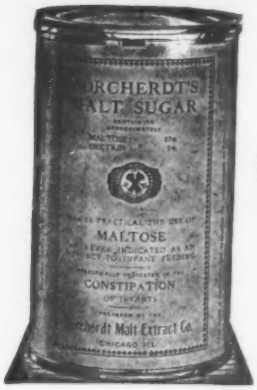

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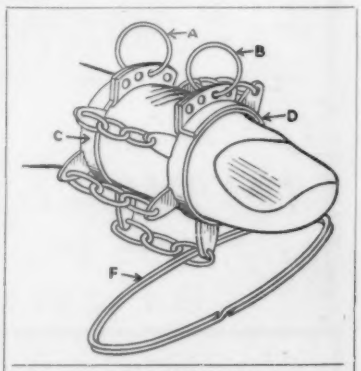

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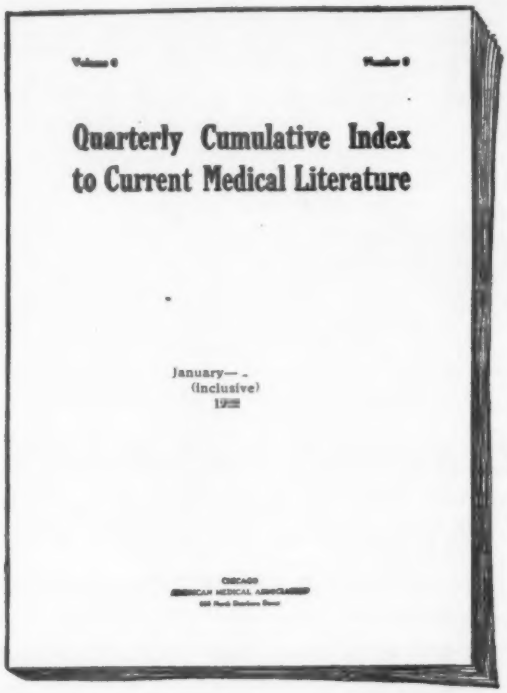

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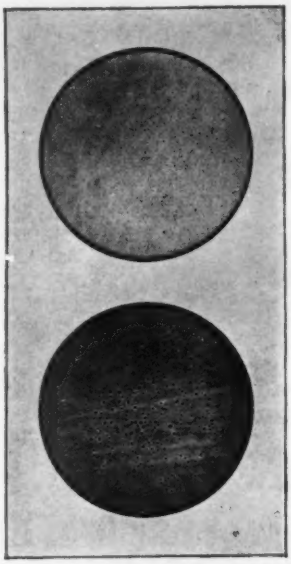

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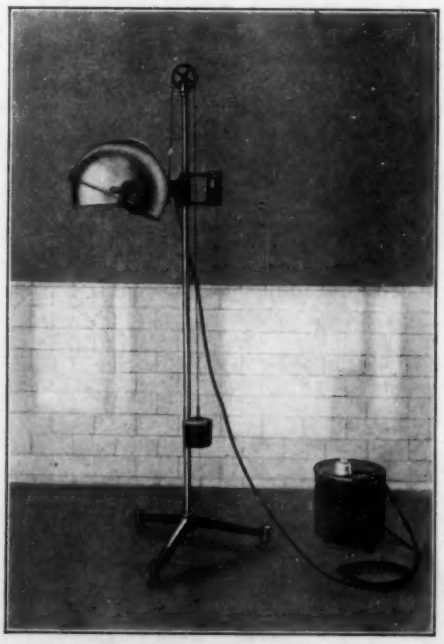

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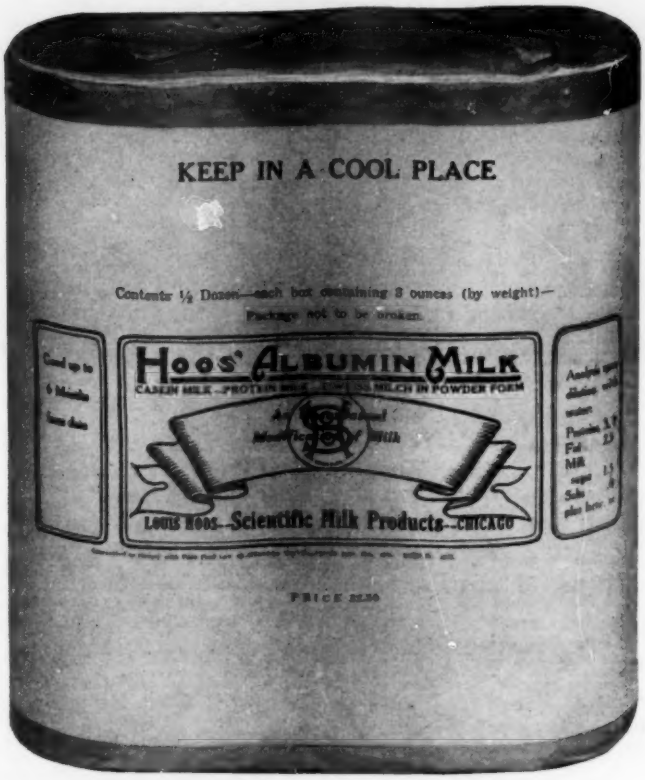

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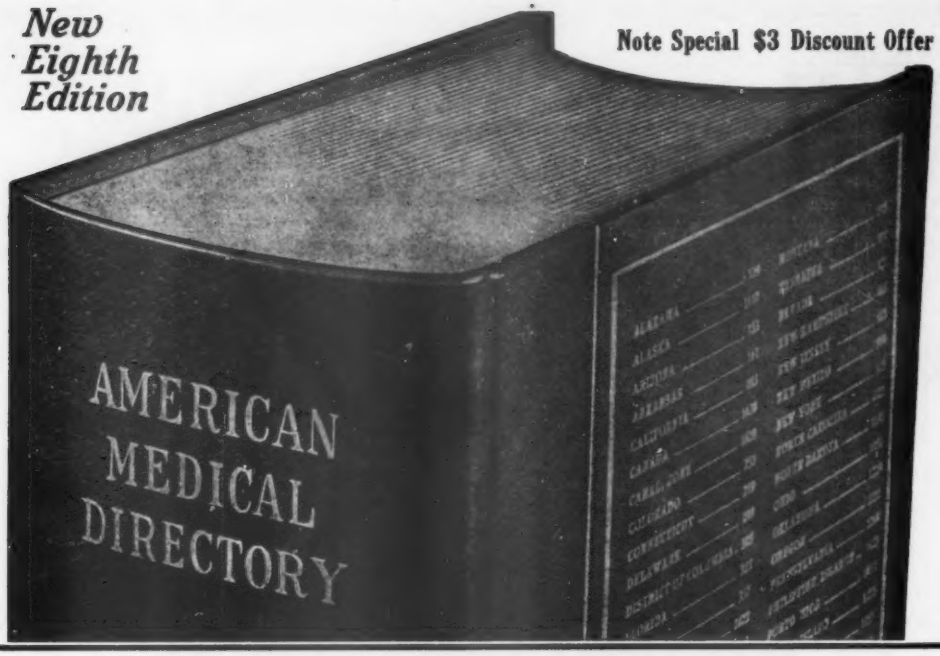

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